A SYSTEM OF MEDICINE

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BY MANY WRITERS

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EDITED BY

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PREFACE

In issuing the seventh volume of this work, and announcing yet another to conclude it, I am exceeding by two volumes the space of our former estimates. Estimates based upon the proportionate parts of the previous English treatises on medicine, even of the more recent of them, proved erroneous in the present phase of extraordinary movement and expansion in our art and in the sciences ancillary to it.

Moreover, when in consultation with the writers of the sections on Diseases of the Larynx, I had to decide whether these departments of medicine should be dealt with as completely and as intimately as experts in the subjects would require, or whether they should be handled in the lighter and more succinct fashion which might prove sufficient for the general practitioner. I decided to accept the former alternative; and, in doing so, I implicitly decided that the special departments of Tropical Diseases, of Mental Diseases, and of Diseases of the Skin should be dealt with on a similar scale, and in respect of no lower a standard.

It is a great satisfaction to me to find that in private conference and in the columns of the press my brethren of the profession, both in Great Britain and in America, have given their cordial approbation to my enlargement of the work so far as to make the reader independent of special handbooks within the sphere of medicine strictly so called.

Even in a pecuniary sense, then, I trust the purchasers of this work will find themselves not the poorer for a present larger outlay.

In the eighth volume the Diseases of the Nervous System will be finished: and the full sections, as already referred to, on Mental Diseases and on Diseases of the Skin will be added. These parts are in revised proof, and will be published forthwith.

My readers will hear with satisfaction that in the editing of these sections respectively I have had the co-operation of Dr. Savage and of Dr. Payne, to whom I take this occasion to offer my cordial thanks for their valuable counsel and aid. To my accomplished colleague Dr. Rolleston I must once more acknowledge my debt, and express my gratitude for his help throughout this large undertaking now so near its fulfilment.

Above all, my thanks are due to my contributors, many of them my personal friends, all my loyal and potent allies. If our correspondence were occasionally enlivened by passing asperities concerning dates and promises—asperities too often endured with a resignation I could have regretted—I can never forget my happy experience of the trust and kindliness which prevailed in every one of them to the forgiveness of criticisms always frank, often no doubt hasty and unreasonable; or of ruthless handling of important manuscripts which in the course of his functions the Editor felt obliged to carry out according to his lights, undeterred by the inward voice which continually reminded him of his own fallibility.

To the Publishers I am grateful for their unfailing courtesy, and for their vigilance in anticipating my own wishes and those of my contributors; also for their submission to the delays which in such undertakings as the present seem inevitable.

Yet to appreciate these aids and encouragements is to bring home to me the responsibility for many shortcomings.

T. C. A.

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cerebral vessels, and then chronic encephalitis. In a similar analysis Dana found only thirty-nine autopsies reported with sufficient fulness to be of value. In nineteen of these, careful microscopical examinations were made; the lesions found in sixteen were pronounced hyperæmia, punctiform hæmorrhages, perivascular exudation, foci of softening, and, in some instances, emboli; all of which changes were most marked in the basal ganglia.

Gross lesions are met with exceptionally, as, for instance, softening consequent on plugging of a large cerebral artery with consequent hemiplegia during life. In an instance recorded by Gray the vertebrals, basilar and middle cerebrals were all blocked. So, too, large hæmorrhages may occur, as in a case recorded by Dr. Bevan Lewis, in which the hæmorrhage was in the cerebellum; and one by Baxter, in which it was in the cerebrum; in both of these cases the apoplexy was fatal.

Discrepant results have been obtained on examination of the motor neurons of the cerebral cortex. Dr. Charlewood Turner, in five cases, found some of the large pyramidal cells swollen, and their protoplasm cloudy and dense-looking. Berkeley, in a case of Osler's which he examined, found no special changes in these motor neurons. Dana, on the other hand, found the pyramidal cells in a state of hyaline degeneration; but his case was complicated by chronic leptomeningitis. Changes in the neurons in other parts of the brain have also been described; thus Meynert found swelling and hyaline degeneration in the cells of the central ganglia, and Elischer similar changes in the claustrum and island of Reil.

Peculiar round hyaline bodies, concentrically laminated and strongly refractile, to which the name "chorea-corpuscles" has been applied, have been found in the perivascular sheaths of the vessels of the corpora striata and internal capsule, and have been described by Elischer and Jakowenks; but Wollenberg, who carried out control investigations in forty-six brains of persons not affected with chorea, beside the brains of six persons so affected, concluded that, though these bodies are present in some cases of chorea, they have no special significance, as they are also to be found in persons who have never had this disease.

The changes which have been found in the pons medulla and spinal cord are no more characteristic than those already described as sometimes met with in the brain; they consist in hyperæmia, punctiform hæmorrhages, and perivascular round cell exudation. The supposed degenerative changes described by Elischer in the cells of the neurons of the spinal cord are in no way characteristic; and Berkeley, in the case which he examined, failed to detect any changes in these cells, or in those of various nuclei in the medulla. An instance of hæmorrhage into the central canal of the spinal cord has been recorded by Steiner.

Meningitis, either cerebral or spinal, has been met with as a complication in exceptional cases.

The changes described in the peripheral nerves by Elischer—hyaline swelling of the axis-cylinders, with increase of interstitial tissue—are of questionable significance; but Frey has found changes characteristic of multiple neuritis.

Several observers have attempted to isolate micro-organisms in chorea, but the investigations of Pianese seem the most complete. He has obtained from the nervous system of the subjects of chorea a bacillus which he has cultivated on artificial media, and which, when inoculated into animals, produces convulsions. He has further obtained pure cultures of the same organism from the central nervous system of the animals inoculated. Maragliano, however, regards this microbe as probably the Bacillus coli, and considers that a coccus also found by Pianese was probably the more potent agent, as he considers the staphylococcus the chief source of infection in chorea; this microbe has been found twice as often as all the other organisms put together.

Rod-like bodies in the tissues have been described by Donkin; cocci in the blood by Richter and by Triboulet; a diplococcus in the meninges by Dana; a cladothrix in the meninges and cardiac vegetations by Naunyn; and S. pyogenes aureus, in the latter situation and in the parotid gland, by Berkeley. Apert found a diplococcus similar to that found by Triboulet in acute rheumatism; it was obtained from the blood of a chorea patient, but (apart from local induration) inoculation of the diplococcus into the guinea-pig was negative. Triboulet and Hoctrovi have both found staphylococci in the blood during life.

Pathology.—Does chorea fall out under a variety of circumstances, owning no indispensable cause? From the widely different etiological factors it might seem that such is the case; but on closer scrutiny we find that these diverse factors may be grouped in three classes. The first may be unstable conditions of the nervous system, of congenital origin, or induced by developmental changes; the second, similarly unstable conditions of the nervous system induced by accidental causes, such as shock or great emotion, acute illness or overwork, mental or physical; and in the third class we may place the immediate causes of the particular attack. We have seen that the phenomena of chorea may be due to some toxic agent; or, on the other hand, they may be evoked in a reflex manner through some source of peripheral irritation, such as eye-strain. Yet, however effective a contingent cause, such reflex influences cannot possibly generate a disease of which the nervous phenomena form but a part; unless the endocarditis in chorea results from an altered blood state brought about by excessive muscular action—a position which is indeed untenable, for the endocarditis may precede the muscular spasms, or may occur in cases in which these are but slightly marked. Moreover, we know of no other affection attended with excessive muscular action in which endocarditis occurs. Far more potent factors are defective nutrition, bad hygiene, and the like, which may aid in the generation of a toxin in the body, or favour its action if introduced from without. There is much that suggests that chorea is generated by some toxic agent, either the result of an altered blood state or of some infective agent introduced into the system from without. The close association between chorea and rheumatism suggests also that some toxic agent is operative in both cases; and it was formerly supposed to be a chemical one, depending on such altered blood state, rather than the product of any organised virus. But the modern tendency is to regard acute rheumatism also as the result of micro-organismal infection; and Sahli, in support of this view, instances the acute onset, the definite course of the disease, its resemblances to a septicæmia, and the common association of arthritis with other infective processes. Several forms of micro-organisms have been described in acute rheumatism; and in some cases Sahli found an organism of but slight virulence, apparently identical with the S. citreus.

The points that suggest that chorea may be the result of an infective process are as follows:—endocarditis is admittedly a morbid state characteristic of an infective process, and we have already seen how commonly this condition of the heart is met with in chorea; the relation of the disease to age and season is similar to that observed in connection with other infectious diseases; and the clinical manifestations of a well-marked case of chorea resemble in all respects those of diseases of known infective origin.

So that both in rheumatism and chorea there is much to suggest that the manifestations may be due to an infective process; but in connection with neither disease can it be said that we have sufficiently trustworthy bacteriological data on which to found such a doctrine. If the infective origin of these two diseases be hereafter satisfactorily proved, there will be little difficulty in understanding the close relationship which undoubtedly exists between them. It would not be necessary to assume that the same poison is operative in both cases, but simply that the poison responsible for the nervous manifestations of chorea, being of an infective nature, is capable, in common with other infective agents, of setting up arthritis and endocarditis. This brings us to the interesting question raised by Flexner and Barker, who suggest that rheumatism itself will be found to depend, not on any single virus, but on a variety of different infections by pyogenetic organisms. Be this as it may, it is clear there is no necessity to assume that rheumatism and chorea are dependent on one and the same infective agent; on the contrary, the evidence is opposed to such a view, unless we assume that neither disease is the result of a specific organism, but that both depend on a mixed infection; and that whether the joints and heart, or the nervous system and heart, bear the chief brunt of the attack depends on the numerical relations of the different organisms in any given case. In other words, it may be that in such a mixed infection organisms whose toxins have a deleterious effect on the synovial membranes of joints, and others which are capable of exerting a baneful influence on the nerve-centres, effect an entry into the system at the same time; and that acute rheumatism or chorea is determined as one microbe or another is present in greatest abundance, or in most virulent form.

It seems scarcely necessary for us to do more than note in passing that the doctrine formerly advanced to explain the relationship between chorea and rheumatism attributed the former to cerebral emboli consequent on endocarditis. Such an explanation necessitates the assumption that endocarditis always precedes the nervous manifestations of chorea, whereas very few instances of this sequence have been met with; moreover, the nervous manifestations have resulted in death without any endocarditis having been afterwards detected.

Fascinating as is the hypothesis of the infective origin both of acute rheumatism and chorea, it is far from proved: and the same may be said of our knowledge of the affinities of the two diseases. There are many facts in the causation of chorea which demand careful consideration before we hastily accept any such generalisation. That such remote antecedents as temperament, age, sex, season, and other debilitating influences affecting the nervous system, may favour an infective agent, is in keeping with what we know of infective processes generally; but how are we to explain by this hypothesis those instances in which the manifestations of chorea have followed so closely on fright that it seems impossible to escape from the assumption that we are dealing with cause and effect? That fright is capable of producing a profound effect on the motor elements of the nervous system is clear from many considerations; and there is no difficulty in understanding how great this effect may be when fright is operative in persons whose nerve-centres are in a state of instability, whether congenital, evolutionary, or otherwise induced.

Symptoms.—In chorea a disordered condition of the motor centres is evidenced by the involuntary spontaneous movements, and by the defective power and precision of voluntary movement. Each of these defects is seen in different degrees of intensity and in various combinations in different cases. The first indication often is that the patient is always fidgeting, though the movements are so similar to those seen in nervous children without chorea, that they may fail to suggest any other interpretation. These movements usually begin in the arms or face, and exceptionally in the legs. Thus various grimaces and contortions are seen; at one moment the eyebrows are raised, at the next an eye is closed and then opened, the upper lip is drawn upon one side and then falls again, the angle of the mouth is drawn to one side, or the mouth is first opened then closed—all of which movements are carried out in a purposeless manner. Or it may be that the head is suddenly twisted to one side, or jerked back; or the patient shrugs one shoulder; or the whole arm is jerked forward from this joint. So, too, when sitting down with the hands on the lap, the hand may be resting naturally when it is suddenly turned palm upwards, and as abruptly back to its original position; or the fingers are irregularly extended and flexed without purpose. Implication of the trunk muscles causes writhing movements, or swaying from side to side, as the patient sits or stands. When the legs are affected the momentary contractions of their muscles cause jerking of the trunk first to one side and then to the other; or in walking the leg is rotated, it may be first in and then out; or one leg is thrown across the other, as if about to become entangled with its fellow.

The spasmodic movements are sudden, and each is of very short duration; they may be simple or complex, and are always irregular, both as regards the degree of resulting movement and as regards the intervals at which they are repeated. At first the patient may have to be closely watched before the movements are noticed; but later they require no such close scrutiny for their detection: so, too, the intervals between the spasms are much longer at first, and become shorter as the disease progresses, until there may be scarcely a single moment during the patient's waking hours when some part of the body is not in movement. Mental excitement and any attempt to perform a voluntary movement aggravate the spasms; while mental and physical quietude diminishes, and sleep, whether natural or artificially induced, arrests them; except in rare instances of very severe spasms, in the majority of which cases sleep is prevented. According to Jaccoud, in less severe cases the spasms sometimes remain in abeyance for a little time after the patient wakes from sleep. Weir Mitchell and Rhein find that in some cases there may be severe choreiform movements during repose which entirely disappear on some muscular act being performed. Dr. J. W. Russell has recently confirmed these observations, and has, moreover, called attention to the fact that the power of inhibiting the choreiform movements can be well demonstrated by making the child attempt to write; then it will be seen that the patient can, for a time, inhibit choreic movements of the most pronounced kind, and write, it may be, even a word composed of six or seven letters with perfect steadiness. Like Weir Mitchell and Rhein, Russell divides cases of chorea into certain groups according to the character of the handwriting:—(i.) the writing gives evidence of power of control over the movements, incoördination being probably present in many of the cases; (ii.) the power of control is practically complete and there is no incoördination; (iii.) choreic movements are either absent or very slight, and yet much incoördination is revealed by the handwriting; (iv.) control over choreic movements is perfect, but the handwriting betrays great incoördination; and (v.) choreic movements are moderate, but writing impossible from mental defect.

According to von Ziemssen, all the movements are not involuntary, some being voluntary and quickly added for the purpose of concealing an immediately preceding involuntary movement.

We have seen that all degrees of severity of the muscular contractions are met with; and not only may the most grotesque grimaces be constantly occurring, but the ocular muscles may be involved, the eyes turning concomitantly with the head and preserving their parallelism, or resulting in squint when the degrees of spasm in the muscles of the two eyes are unequal. The globes return to their normal position, or are jerked into some other abnormal position.

In a case recorded by Dr. Tuckwell, so powerful was the spasm of the muscles which raise the lower jaw that several teeth were broken. The head may be banged against adjacent objects, and the limbs greatly bruised as they are violently thrown about. The arms are always affected in greater degree than the legs; the jerking movements may be so severe as to make it quite impossible for the patient to effect any purposive movement with the arms: thus in attempting to raise a glass or cup to the lips the arm is thrown in various directions before the vessel reaches its destination, and most of its contents are probably spilled before it is grasped by the teeth and the remains of its contents gulped down in the most hasty manner possible.

When the trunk muscles are involved their spontaneous contraction may be so powerful as to throw the patient out of bed. Similarly voluntary movements of the lower limbs may be so interfered with by the muscular contractions that walking may be made quite impossible. In cases of moderate intensity the spontaneous movements may be limited to one side of the body; but Dr. Hughlings Jackson has shown that this is true only of the limbs, the muscles of the face and trunk being always bilaterally affected. In these cases of hemichorea Dr. Jackson is of opinion that the right side is the more frequently affected: according to Jaccoud, it is the left, and the figures quoted by Dr. Pye-Smith and Sir William Gowers both show a small balance in favour of the left side. The movements may remain limited to one side or part of one side throughout the attack; or the opposite side may be attacked, with or without cessation of the spasm on the side first affected; so, too, movements involving the arm and leg on one side may spread to the opposite leg without implication of the corresponding arm.

But, as was stated at the outset, not only may voluntary movement be interfered with by these spontaneous movements, but two other factors may be in operation: an incoördination or want of precision in the performance of voluntary movements, or an actual want of power due to muscular weakness. In the first of these cases, instead of some jerky spontaneous movement being the first to attract attention, this failure to co-ordinate movement may be noticed first: the motor centres do not obey the dictates of the will, so that when the patient tries to relax some set of muscles, for the due completion, it may be, of some muscular act, there is a marked delay which of course seriously interferes with its efficacy. Or the motor centres may bring about relaxation of a group of muscles during the performance of some voluntary act without having received any command to do so, and oftentimes with disastrous results, as objects in the hand may be suddenly dropped and broken. The incoördination of movement may be shown in a different way, the patient over-shooting the mark in the attempt to lay hold of or pick up some object.

It will be readily understood that the spontaneous movements and the incoördination of movement may in any given case be so combined as to make it difficult to distinguish the operative factor; for, of course, a voluntary act may well be interrupted, not in consequence of the sudden unwilled relaxation of a group of muscles, but of the unexpected intrusion of some spontaneous movement. Voluntary movements are irregular, and are performed in a sudden and spasmodic manner; partly on account of the involuntary movements, and partly to dodge them. If the patient be told to protrude the tongue the act may be delayed in accomplishment, but the organ is then shot out of the mouth and as suddenly withdrawn, perhaps just in time to escape the snap of the closing jaws. In severe attacks deglutition is much interfered with. Speech is often affected, words are spoken rapidly, and in an explosive sort of way, the sentence being frequently interrupted by a hasty inspiration which may clip off the last part of the word which immediately precedes it; or there may be an inspiration of this kind after each word that is uttered; or a word can only be uttered spasmodically in parts. Owing to the spasmodic movements of the tongue and muscles of articulation, speech may indeed become quite unintelligible, or the patient may be deterred from making the attempt. Von Ziemssen, on laryngoscopic examination, has observed irregular movements of the vocal cords; and that the tension of the vocal cords is defective is evidenced by the fact that a note cannot be sustained for any length of time; moreover the voice is low pitched and monotonous.

The movements of the thorax in respiration are also made disorderly, chiefly owing to implication of the diaphragm. Inspiratory efforts do not occur in any regular order, either as regards time or depth of movement; thus irregular pauses intervene, and one inspiration is deep, perhaps excessively so, while another is shallow or abortive. The incoördination of respiratory movements sometimes shows itself in an alternation between abdominal and thoracic respiration. Romberg refers to two cases in which a whistling or snapping sound accompanied inspiration; and in one of them spasm of the intercostal muscles caused sudden drawing in of the thorax. The same observer speaks of a case of troublesome choreic hiccough. Apart from any independent cardiac affection, the heart's rhythm may be made irregular by the disorderly respiratory movements.

A third factor which may interfere with voluntary movement is muscular weakness, a phase of the disease which was clearly recognised by the earlier writers on the subject. As a rule, this loss of power is most marked in cases in which there is little if any spasm, when it is commonly of hemiplegic distribution. Though the weakness amounts to definite paresis there is never anything approaching complete loss of power. Todd fully described this type of the disease, to which West applied the term "chorea mollis," and for which Sir Wm. Gowers has proposed the name "Paralytic chorea." This element of muscular weakness probably accounts for the slovenly way in which some choreic patients shuffle their feet along the ground in walking. In twenty-eight of Osler's cases, in which muscular enfeeblement was noted, it was monoplegic in sixteen, the arm being affected in each instance; paraplegic in six; and hemiplegic in four: in one case all four extremities were affected, and in one both arms without the legs.

The weakness may come on gradually during the course of an attack of chorea with spasms; or it may come on in a similar manner or quite suddenly before any spasms have been detected; in a third group of cases the paresis is noted after the spasms have ceased. When spontaneous movements and paresis both exist they usually disappear together; but in some instances the paresis persists, it may be even in considerable degree, for a long time after the cessation of the spasmodic movements. Osler speaks of a case in which wrist-drop persisted for two years after an attack of chorea of the right arm, with which it came on.

Electrical reactions.—These have been studied more especially by Benedikt, Rosenthal, Schmitt, and Sir Wm. Gowers, all of whom are agreed that in some cases electrical irritability may be increased, both in the nerves and muscles; and to both galvanism and faradism. So, too, an altered qualitative mode of response to galvanism has been described, in which the anodal closure has resulted in contraction of the muscles with as weak a current as the cathodal closure, instead of the response being more readily obtained with the latter than the former, as obtains normally. Thus KCC = ACC, instead of KCC > ACC.

Sensory symptoms.—Headache is common; it may precede the spontaneous movements, or appear later; and is either more or less continuous or is paroxysmal. Sir Thomas Watson met with some cases in which the pain was limited to the side of the head opposite to the affected limbs.

Pain in the nerves and muscles of the affected limbs is rare, and, of the exceptional instances in which they have been described, most of the cases have been hemi-chorea, and have been designated "painful choreas" by Weir Mitchell. Instances have also been recorded in which pain has occurred in parts subsequently involved in the spontaneous movements. The nerve-trunks are said in some cases to be tender on pressure, and tender points have been described along the spine at the points of emergence of the spinal nerves from deeper structures. Cartier, Triboulet, and Marie have especially insisted on these, and it has been said that they are limited to the affected side in hemi-chorea; however, such phenomena, though carefully searched for by Osler in a large number of cases, were very rarely met with; and there can be no question that in the large majority of instances chorea is an affection unattended by pain. Trousseau insisted on the occurrence of tingling and formication in the affected parts, and others have described similar paræsthesiæ and numbness. The latter symptom Fagge found not uncommonly noted in cases at Guy's Hospital.

Diametrically opposite opinions have been, and still are held with regard to the sensibility in chorea. Most observers are of opinion that if there be any impairment of sensibility it is probably a hysterical manifestation; but Féré asserts that blunting of sensibility is common, and goes so far as to say that this has been found in almost all cases in which sensibility has been carefully studied, especially in parts where the spasms are most pronounced. Sir Wm. Broadbent is also of opinion that sensibility is frequently diminished, and Dr. Purves Stewart detected it in ten out of forty-one cases.

Reflexes.—Discrepant statements are made by different authors on this subject; some consider that the reflexes are usually normal, others that they are usually exaggerated; and others again that the superficial reflexes are commonly diminished. My own experience is that the reflexes are usually normal; but in some cases there may be slight increased activity of the tendon jerks, in others, and more especially in the paralytic variety of chorea, they may be diminished in varying degree up to complete abolition; in some cases the resulting contraction of the quadriceps extensor is unduly sustained. In 50 cases of chorea, examined by Sinkler with the object of ascertaining the state of the reflexes, the knee-jerks were found normal in 26, diminished in 15, and absent in 9.

Sphincters.—The sphincters are not affected, though in severe cases the evacuations may be expelled involuntarily; and a similar result may be met with in cases in which there is much mental hebetude present.

Trophic disturbances.—There is no spontaneous tendency to bedsores; but the incessant rubbing of certain parts against the bed in severe cases and self-inflicted cutaneous abrasions may lead to their formation. Such sores are especially apt to form in advanced stages of severe cases, when general nutrition is much lowered, and when, as often is the case, mental hebetude is so great that the evacuations are passed under the patient.

Mental condition.—The psychical disturbances in chorea have been studied especially by Marcé. Though bright and intelligent at the outset there are few patients in whom there is not some mental affection, though in the majority it is fortunately slight. The alteration may be in the form of diminution of attention, mental dulness, and loss of memory; not only is the child unable to learn lessons, but it no longer cares about story books, and the face wears a dull, heavy look, singularly wanting in variety of expression. Or there may be marked perversion of character; the child becomes irritable and whimsical, is liable to emotional attacks, and does all sorts of odd things. Then again, there may be hallucinations (Axenfeld and Huchard), most commonly of sight, though sometimes of one of the other special senses; or it may be of common sensibility, or under rare circumstances even of the genital sense. Such hallucinations may be originated in dreams and perpetuated during the waking hours. The degree of mental change bears no relation to the severity of the other symptoms; and the symptoms may progress to dementia, melancholia, perhaps with suicidal intent, or to mania in which the patient jabbers incoherently, shouts, and sings. It is to this class of cases that the name "chorea insaniens" has been applied; in many such patients sent to asylums the physical condition is entirely overlooked. The delirium is usually recovered from in acute chorea; but where there is any hereditary taint of insanity it may be the starting-point of progressive and permanent mental deterioration.

Convulsions are rarely met with in the course of chorea, and epileptiform convulsions of unilateral distribution still more rarely; nevertheless, originating in this way, convulsive attacks have been known to persist, and to constitute ordinary attacks of epilepsy. Certain peculiarities of the convulsions have been noted in some cases—such as an admixture of choreiform spasms in the attacks, or an associated condition of cataleptic rigidity.

Ocular phenomena.—In addition to the transitory squints and the like, which result from irregular spasms of the eye muscles, the following ocular phenomena also may be met with. The pupils are frequently dilated, and this has been observed to be more marked on the side on which the spasmodic movements have been most severe. The optic discs are usually normal; but evidences of slight neuritis are met with exceptionally, and as hypermetropic errors of refraction have been also present in many such cases, it has been supposed that the hypermetropia may be responsible for the changes in the discs; but that it is not the only factor in the production of the change is proved by the disappearance of the neuritis without correction of the hypermetropia. This condition of the optic nerves lends support to the view that chorea is of infective origin, for it is more probable that the neuritis depends on some blood state present than that it is secondary to an intracranial lesion.

Circulatory system.—The pulse is frequently rapid, especially where the spontaneous movements are severe; in some cases it is irregular, according to some observers, owing to a veritable chorea of the heart; while others regard it as depending on the disturbance of the respiratory movements of the thoracic cage. Sir Wm. Gowers has observed several instances in which posture had less effect than in health, there being little difference, if any, between the pulse-rate in the upright and recumbent postures.

The most important defect in the circulatory system, however, is in the condition of the heart itself. Apart from rapidity and, possibly, irregularity of action, murmurs may be heard over the precordial area the significance of which has been much discussed. Three explanations have been offered for these—(*a*) that they are hæmic, depending on associated anæmia; (*b*) that they are the result of regurgitation at the mitral orifice consequent on irregular contraction of the papillary muscles not closing the mitral flaps effectively; (*c*) that they are due to organic valvular lesions. The second of these explanations is one about which little need be said, in that we have no means of proving or disproving it. With regard to the other two there can be no question that both are operative in different cases, or it may be even in the same one. While it may be difficult in some cases to be certain which of these two factors is responsible for the murmurs, as a rule the following considerations will lead to a correct interpretation. In the first place, doubt can only arise where the murmur in question is systolic in time; a presystolic or diastolic murmur being unequivocal evidence of organic disease. I cannot concur with those who hold that diastolic and even presystolic murmurs may be hæmic in origin. In the next place, it is comparatively rare for organic disease to be found at the aortic orifice in chorea, the mitral valves, as we have already seen, being affected in the large majority of cases. In anæmia the most common murmurs have their seat of maximum intensity at the base; or if, as sometimes happens, the point of maximum intensity is in the fourth intercostal space close to the left border of the sternum, it is heard up to but not beyond the apex of the heart; whereas in the organic cases by far the most common murmur is a systolic one having its seat of maximum intensity at the apex and conducted into the axilla, and it may be round to the angle of the left scapula. The presence of a murmur at the apex conducted into the axilla may, however, depend on dilatation of the left ventricle in anæmia, without the presence of organic valvular disease; so that it is necessary to take other points into consideration before arriving at a conclusion, such, for instance, as other evidences of anæmia in the patient, the presence or absence of a hæmic murmur at the base having its point of maximum intensity in the second left intercostal space, the presence or absence of a venous hum in the neck, and so on. But as organic valvular defect and anæmia may coexist, some cases may have to remain unsettled until the anæmia has been removed. Even this does not entirely settle the question, however, for, as Fagge has insisted, even murmurs observed in rheumatism may similarly pass away; Kirkes, Wilks, Baxter, and Frank have all recorded cases in which no murmur was present during life, and yet vegetations were found on the valves after death.

Apart from rapidity and possible irregularity of action, there may be definite organic disease of the heart. According to Sir Wm. Gowers, in nine out of every ten fatal cases of chorea, disease of the valves of the heart is found. Sturges collected 80 cases from four of the large London hospitals, in only 5 of which were the heart and pericardium free from disease. Of 34 cases collected by Raymond, in addition to the cases reported by Sturges, the condition of the heart is only given in 19, in all of which there was endocarditis. Osler has since collected from recent records 73 cases with endocarditis in 62 of them.

Murmurs indicating the existence of organic disease of the heart may be present before the attack of chorea, and may then be due to a previous attack of this disease, or of acute rheumatism. On the other hand, primary attacks of chorea may be met with in which there had been no antecedent rheumatism, and in which no murmur is present. In some such cases a murmur may appear during the attack, and may persist after the patient is convalescent; while in other instances, as the patient recovers from the chorea, the murmur may disappear. Another class of cases is met with in which no murmur is heard throughout the attack, but presents itself at some subsequent period.

The most common murmur is a systolic one produced at the mitral orifice; but sometimes, where the murmur existed before the onset of the chorea, a presystolic murmur is heard alone, or in combination with one systolic in time. Murmurs indicating the existence of aortic disease are much more rare, as may be judged from the fact that the Collective Investigation Committee, previously referred to, found 116 cases of mitral disease as opposed to only 6 aortic cases. So, too, Sir William Gowers states that out of 250 cases of chorea he only met with 2 cases of aortic regurgitation; and Osler, out of 72 in which there was evidence of organic disease of the heart two years and more after chorea, found 4 cases only in which the aortic valves were affected.

With regard to the association of pericarditis with chorea we have already seen that Bright, and some others of the older physicians, especially at Guy's Hospital, recognised this, and that Bright founded a hypothesis of chorea on this association. The pericarditis is usually associated with endocarditis, though it sometimes occurs alone. Thus of 21 cases of chorea occurring in acute rheumatism Sibson found that 15 had pericarditis, 14 endocarditis, 3 doubtful endocarditis; 6 had no pericarditis, and 3 no endocarditis. So, too, of 73 recent autopsies in chorea, collected by Osler, there were 19 cases of pericarditis, in only 2 of which endocarditis was absent.

Cutaneous affections.—The more important of these serve as another link in the chain of evidence in support of the close affinity between chorea and rheumatism. Erythema nodosum, arthritic purpura, purpuric urticaria, and subcutaneous fibrous nodules belong to this class. Herpes zoster is sometimes seen, but probably owes its origin to the arsenic which is administered to such patients. Excessive pigmentation of the skin, when present, is probably always due to the same drug; but absence of pigment in the skin in patches has been described. So, too, alopecia areata is said to have been observed; bald patches on the head, the result of the incessant rubbing against the pillow, must not, however, be mistaken for the baldness of alopecia.

Urine.—An excess of urea was found by Walshe and others; and, according to Todd, uric acid may be deposited in considerable amount. Dr. Handfield Jones has described a similar increase of phosphates. These conditions of the urine have been found proportional to the severity of the attack of chorea. The pigment urohæmatoporphyrin, discovered by Dr. M'Munn in the urine of rheumatic subjects, was found by Dr. A. E. Garrod in the urine of fourteen out of twenty cases of chorea; this he regards as additional evidence of the close relationship between rheumatism and chorea. Albumin, when present, usually indicates nephritis; according to some observers, it may be produced by renal embolism also; of this statement, however, satisfactory proof is wanting. Glycosuria has also been recorded.

Temperature.—The milder forms of chorea may run an apyretic course; and even when the movements are severe no elevation of temperature may be detected. Other cases present a slight degree of pyrexia, irrespective of any detectable complication; there may be a rise of a degree or two in severe cases, and in chorea insaniens a temperature of 104° F. may be reached. Elevation of temperature may, however, occur as a result of some complication, usually arthritic or cardiac; and, though a temperature of 105° F. has preceded death where no complication was detected, hyperpyrexia is usually the result of some complication, and even then in chorea is a rare event.

Recurrence.—A patient who has once had an attack of chorea is liable to be similarly affected on one or more subsequent occasions. This tendency of the disease to recur was known to Sydenham, and has been insisted on by all subsequent writers. Sir William Gowers found that a third of his patients had more than one attack of chorea, and he mentions one in whom there had been as many as nine attacks. Osler's statistics show that, out of 410 cases, there were two attacks in 110, three in 35, four in 10, five in 12, and six in 3.

The interval between a first and a second attack of chorea varies from a few months to several years, but is most commonly about a year. Instances in which recurrence has been reported within a few weeks of the original attack ought probably to be regarded as relapses of an attack whose course is not fully spent, rather than as true recurrences. The effect of season on the recurrence of chorea has been noted by a good many observers, but notably by Weir Mitchell. Such fresh attacks are most commonly met with in the spring; but instances of their occurrence in the same subject during successive autumns have also been observed. Rheumatism does not appear to have any influence on recurrences; though Osler reports a higher percentage of arthritic changes in cases of three attacks or more than in those of one or two attacks. As the heart has been more frequently affected in recurrences than in primary attacks, some have supposed that cardiac disease favours such recurrences. It is far more probable, however, that the true explanation of this association is that, as endocarditis tends to occur in chorea, the greater the number of attacks of chorea the more likely is endocarditis to be found. Indeed, evidence of the occurrence of endocarditis during a recurrent attack has been met with in cases in which there were no signs of it at the beginning of the attack.

Females are more prone to a recurrence than are males in about the same proportion as they are more liable to original attacks; but in cases of recurrence there is an increasing tendency for the number of females affected to preponderate over the males; so that in cases of four or more attacks the subjects are nearly always females.

In many cases no cause for the recurrence can be detected; in some it has been ascribed to fright. The relationship between a fright and the onset of the symptoms of chorea is in some such cases so close as to compel us to regard them as cause and effect (p. 836); but it is equally certain that in many cases no evidence whatever of fright can be obtained. Overwork at school has been also regarded as a cause of recurrences. On the relations between pregnancy and chorea sufficient has already been said to show us that the former condition is a potent factor in exciting second and, more rarely, third attacks of chorea in women; and that, exceptionally, pregnancy may be responsible for several recurrences.

As a rule the manifestations of the disease in second and subsequent attacks are not so pronounced as in the first; but many notable exceptions occur in which the symptoms in a recurrence are much more severe than in the original attack. The parts affected by the spasms may be more or less the same in recurrences as in the primary attack; or there may be no such correspondence, the spasm being either more widespread than in the first attack, affecting totally different groups of muscles, or, while involving those originally affected, preponderating in others that formerly escaped. Thus in some cases successive attacks are more or less restricted to the same side of the body, while in others such limitation occurs on the opposite or on the same side indiscriminately; or again both sides may become affected, but the opposite to a much greater degree than that which suffered, it may be exclusively, in one or more former attacks.

Diagnosis.—There is no difficulty in diagnosing typical chorea: the spasmodic movements might, however, be mistaken for those seen in some cases of cerebral disease in infancy; indeed this mistake has probably been made in some of the alleged instances of chorea in very young children. The history serves to distinguish the two conditions; chorea is usually of recent onset, while the cerebral condition is one of earliest infancy.

So-called "paralytic chorea" may be very difficult to recognise with certainty; spontaneous movements may be almost entirely absent, and thus the case may come to be regarded as some other form of paralysis. As a rule the history suffices to exclude other forms of paralysis, for the weakness in chorea usually comes on gradually in the course of some weeks. We have seen, however, that there are exceptions, and that considerable paresis may be of sudden onset in this disease. When, moreover, the weakness is hemiplegic in distribution, and there is organic disease of the heart, the diagnostic problem may be one of considerable difficulty; in such cases, however, there is no history of convulsions or loss of consciousness, the paralysis is never so well marked as in occlusion of a cerebral vessel, and moreover, in chorea, even when the leg is affected in conjunction with the arm, the face escapes.

In the case of monoplegic weakness, as for instance of one arm—the most common form of paresis in chorea—the slow onset distinguishes the condition from organic affections: or, if the onset happen to be sudden, the escape of both leg and face and the absence of convulsions and other grave symptoms at the onset exclude an organic cerebral affection; the absence of atrophy and the reaction of degeneration exclude acute anterior poliomyelitis; and they also exclude peripheral neuritis, the exclusion of which may possibly be aided by the absence of any defect of cutaneous sensibility, which is certainly the rule in this class of cases of chorea. Moreover, by carefully watching such patients, very slight spontaneous movements may possibly be detected from time to time, either in the paretic limb or elsewhere; or some such defect may be detected by making the patient hold the arms above the head or straight out in front of her, with or without the tongue protruded at the same time; again, if the child be told to grasp an object with the affected hand, and to keep the hand tightly closed on the object for a little time, she may fail to do so, the muscles involuntarily relaxing in the manner already described as so characteristic of chorea; or, once more, delay may be detected when the patient voluntarily attempts to relax some group of muscles. The editor of this work has told me of a case of paresis of the forefinger of the left hand in a boy of fourteen which proved to be chorea, as was suspected by himself and by an eminent physician to whom he referred the case. The paresis, which came on gradually in a very healthy boy, and was chiefly manifest as a disability of holding the reins of his pony, stood, as a symptom, absolutely alone for many weeks. Then very slight choreic movements and a transient systolic murmur clenched the diagnosis of a case which for a while could only be guessed at on an estimate of probabilities. In a second case, which came under his care, a girl was admitted into the Leeds Infirmary completely or at any rate very deeply paralysed in all four limbs: she lay motionless, but the movements of organic life persisted. This state was said to have come on rather quickly—in a few days. The negative features of the case led to a provisional diagnosis of chorea, and within a week twitches appeared and chorea became unmistakably manifest.

It may be no easy task to distinguish true chorea from the hysterical simulation of it. In cases of the latter kind a history of imitation can often be obtained; the subjects are generally girls about the age of puberty or adolescence; the movements are more sudden, and single muscles contract: furthermore the movements are often rhythmical, whereas in true chorea they are always irregular.

As was said when discussing the symptomatology, in cases with pronounced mental symptoms chorea may never be suspected. This is especially apt to be the case in the maniacal form of delirium in which the choreic spasms, though present, are masked by the mental state. Moreover in some of these cases the chorea spasms cease when the mania is at its height. Acute mania does not usually attack such young subjects, and in it the patients jabber more incessantly than in the delirium of chorea. Careful watch must, of course, be kept for any choreiform movements.

Senile chorea (p. 862) is distinguished by reason of the patient's age at the time when it first manifests itself; by the fact that the spasm is as a rule slighter in degree than in cases occurring in earlier life; by its usually chronic progressive course, often persisting until the end of life; and by the absence of any relation to rheumatism or endocarditis.

Huntington's chorea (p. 859) has, in common with the senile form, the distinguishing features that have just been described; besides which there is a hereditary history, often dating back several generations. Such cases are even more progressive than the senile form; they invariably persist to the end of life, and are commonly attended by mental deterioration.

In electrical chorea (Dubini's disease, p. 864) the spasms are much more sudden and shock-like; and cases of the kind have been met with only in Lombardy and adjacent parts of Italy. The course is progressive; a large proportion of cases end in death, and the more acute are attended with rise of temperature. Epileptiform convulsions are common; and not only does paresis follow, but the muscles waste, and lose their faradic irritability.

Paramyoclonus (p. 888) is distinguished by the fact that the spasms are much more sudden and shock-like; as a rule they affect similar muscles on the two sides of the body, and are commonly restricted to the muscles of the trunk and proximal segments of the limbs; though in some cases they may be much more generalised. This affection is more harmless than chorea, being in no way related to rheumatism or endocarditis, yet more persistent, lasting in most cases for years.

In double athetosis the movements are slow as a rule, are writhing or undulating, and are attended with permanent stiffness of the limbs; there is also, in some cases, an increase in the volume of the muscles. Such patients commonly suffer from convulsive attacks, and as a rule the intellect is defective.

Prognosis.—The prognosis of chorea varies with the age of the patient; in children the large majority, even of severe cases, end favourably; the report of the British Medical Association Investigation Committee showed the death-rate to be about 2 per cent—nine deaths in 439 cases of the disease. According to Anstie, chorea is much more serious after puberty; and the gravity of the affection in pregnancy has already been insisted upon. The majority of the deaths from chorea in pregnancy result from abortion, whether spontaneous or artificially induced; or indeed, in consequence of the extreme debility which commonly ensues in such cases, a natural labour at full term may prove fatal.

As a rule the more pronounced the psychical disturbances the more serious the prognosis; but this is not always so, for patients with marked mental affection may recover completely, while on the other hand the most severe spasmodic movements may be present, and may end in death, without definite mental disturbance. It must be remembered that where there is much psychical disturbance, though there may be recovery from the attack of chorea, progressive mental deterioration may follow, and may be permanent. Yet mental defects have been known to persist for months or even years, and ultimately to pass away. It must also be remembered that epileptoid attacks in the course of chorea may subsequently become genuine epilepsy.

Apart from complications, the two chief perils are exhaustion from want of sleep and the never-ceasing violent movements. Little less important is the interference with the patient's nutrition, and even with the very acts of taking food. Death rarely occurs in a recurrent attack. These points must, therefore, all be kept clearly before us in attempting to formulate a prognosis in any case of chorea.

Not only have we to express an opinion as to the ultimate result, but also as to the duration of the attack; yet nothing is more uncertain than the latter, for whereas in the majority of cases the attack will come to an end in six weeks, in others it may last as many months. First attacks usually last longer than subsequent ones; the more severe the spasmodic movements the longer is the condition likely to persist; and cases of "paralytic chorea," though presenting little or no danger to life, are often most obstinate. Post-choreic paralysis is sometimes very well marked; but, in the absence of evidence of occlusion of a cerebral vessel, complete restoration of function in the affected parts may be confidently expected.

In some cases the spasmodic movements persist, and result in a chronic chorea which does not appear to affect the general health of the patient materially. Such cases are much more commonly met with in males than in females. The hysterical form, which never ends fatally, often persists for a long time.

The presence of endocarditis does not affect the immediate prognosis, for embolic attacks are singularly rare; but it affects the ultimate prognosis in the two following ways. It may lead to serious structural damage of the affected valves and to the train of phenomena which attend chronic valvular diseases of the heart, ultimately terminating in failure of compensation with its usual consequences; and, if a repeated attack of chorea occurs in a person the subject of former heart disease, the prognosis is less favourable in proportion to the gravity of the latter. This, however, is a factor of minor importance in prognosis. It is not a little curious that although chorea and rheumatism are so closely associated, and pericarditis occurs in a fair proportion of cases of chorea, hyperpyrexia is very rarely met with in the latter disease.

Treatment.—It is of primary importance that any possible exciting cause of the affection should be sought for, and combated, if possible, by appropriate treatment. A sedative and calming line of treatment must be adopted where shock appears to have been operative. Where reflex factors, such as errors of refraction, dental irritation, intestinal worms, constipation, phimosis, and the like are present, the particular source of irritation must be removed. In cases of chorea gravidarum the question of artificial abortion or premature delivery may arise, and this procedure, grave as it is, may in severe cases be imperative.

Rest is one of the most important factors in the treatment of chorea. Even in mild cases, occurring in children, it is important that all lessons should be discontinued, and that the child should not be allowed to make any mental effort. So, too, it is well to begin the treatment with a few days of absolute rest in bed; this must, however, largely depend on the effect which it has on the child; for in cases in which bed is irksome the consequent mental depression may more than counteract the good effect of the means. Where such absolute rest is not deemed advisable, much good may be obtained by getting the child to lie on a bed or couch for an hour or two in the middle of the morning, and again in the afternoon; and various means may be devised to keep it interested and amused during these periods. Although more important in the severer cases of the disease, the amount of rest must be regulated on similar lines; yet, if possible, the child should be kept in bed until all the choreiform movements have ceased. In severe cases this rule should have no exception. We have most of us learnt the value of rest in modifying or possibly warding off the endocardial lesions in acute rheumatism, but we have scarcely realised that this element of treatment may be equally important in similarly modifying or warding off such lesions in chorea. Quite apart from the great good which must result to the motor neurons, which are in an exalted state of irritability, there can be little doubt that rest is none the less needed as a prophylactic or curative measure in the treatment of the cardiac manifestations of the disease.

In cases in which the disease is at all pronounced it is well to combine isolation with the rest treatment. We have already seen how highly strung and excitable the subjects of chorea commonly are, so that it will readily be understood how important it is to reduce all mental excitement to a minimum. This can best be effected by keeping the child in a bright airy room with its mother or a sensible nurse to look after it, and to keep it interested and amused. An important question which frequently arises in private practice is whether it is advisable to engage a trained hospital nurse in such cases. In the treatment of adults there can be no question that this is the best plan, but no general rule can be laid down on this point with regard to children; each case must be dealt with on its own conditions. If the mother be not herself highly strung, or of neurotic temperament, and if the child's own nurse is a sensible and soothing person, it is better not to risk the introduction of a stranger; but where, as so commonly happens, the mother is neurotic, and the nurse has no notion how to keep the child quiet and yet amused, it is better, even at the expense of a little disturbance at the outset, to place a carefully selected trained hospital nurse in charge of the child, and to keep all other people away as much as possible. It need scarcely be added that in selecting the nurse attention should be paid not only to her general qualities, but also to her special experience in the management of children.

If the spasmodic movements are at all severe the patient must be guarded against injury to the limbs. The patient should be on a soft mattress, and any adjacent hard structure carefully padded; prominent parts, like the elbows and knees, may be enveloped in cotton wool. When the spasms are so severe as to throw the patient out of bed, a mattress should be placed on the floor in the corner of the room, and the adjacent wall padded with cushions or mattresses. If there be the slightest tendency to bedsore a water-bed should be provided.

Of no less importance than mental and physical rest is an abundant supply of nutritious and easily digested food. Where the spasms of the head and neck are severe, feeding may be attended with great difficulty, and it may be only possible to administer liquids, and these, perhaps, only by a feeding-bottle. The nutrition of the muscles may be improved by massage—a measure which cannot be employed where the spasms are severe, but which is useful in milder cases, and especially in the paralytic forms of chorea.

Owing to the liability to bedsores scrupulous cleanliness must be observed, especially in those patients who pass their evacuations in the bed; there is commonly so much mental apathy in these cases that the patient may be unaware of mishaps, the early detection of which must depend upon the nurse's watchfulness.

When the movements are not too violent, warm baths do good and are grateful to the patients; care must, of course, be taken to prevent any injury by self-inflicted blows. Though satisfactory results have been obtained by the method of producing diaphoresis by antimonial wine, supplemented by the hot air bath, it is not a measure to be recommended in the majority of cases of chorea, in which the degree of debility does not admit of further depressing influences. In the severer forms of chorea attended with high temperature the cold pack is useful; or the cold bath may be substituted where the spasmodic movements do not prevent this.

Two classes of drugs are chiefly useful in the treatment of chorea—tonics and sedatives. Of the former class arsenic has a deservedly high reputation. The general condition of the patient much improves under its use; and, whether the drug have any power of controlling the spasms directly or not, we may surmise that it improves the nutrition of the neuron. Whatever be the true explanation of its action, the fact remains that a large number of cases of chorea do well under its use. The drug is usually given by the mouth, in gradually increasing doses, after meals; three to five minims of liquor arsenicalis may be given three times a day as an initial dose, and the amount increased by one or two minims every third or fourth day until a dose of fifteen minims three times a day is reached, or until the stomach becomes intolerant of the remedy. Owing to the great intolerance of the stomach in some cases, the use of arsenic subcutaneously has been recommended by Eulenburg, Hammond, Wiederhofer, and others—a procedure which is feasible in the treatment of adults, but which is attended with obvious drawbacks in the treatment of children. An advantage claimed for the method is that much larger doses can be given without the intolerance which appears when similar doses are given by the stomach: indeed the administration of the drug subcutaneously is sometimes continued by subcutaneous injection after the gastro-intestinal tract has become intolerant. It must be remembered, however, that such large doses of arsenic as these cannot be given with impunity; for an attack of peripheral neuritis may be the consequence, the treatment thus substituting one disease for another, a state of things hardly to be desired. A much less important effect of the arsenical treatment which sometimes occurs is pigmentation of the skin, an appearance apt to be attributed to an insufficient use of soap and water.

In anæmic subjects of chorea iron may be advantageously combined with arsenic, and if arsenic has to be suspended from any cause, it is a useful substitute. In weakly children the syrup of the phosphate of iron, or some similar preparation, alone or in conjunction with cod-liver oil, is of advantage. Phosphate of lime, cod-liver oil, preparations of malt, and the like, are all of value as adjuvants or temporary substitutes during a course of treatment by arsenic or some other nervine tonic.

Zinc, in the form of either the sulphate or oxide, was formerly much used, the dose being gradually increased up to twenty grains. The valerianate has also been employed, and of late the bromo-valerianate has been recommended.

No good comes of the use of strychnine in the earlier stages or in the acuter manifestations of chorea; but in chronic cases, especially where motor paresis is prominent and the spasmodic movements absent or slight, this drug is often of distinct service.

Good results have been obtained by H. C. Wood with large doses of quinine; a mode of treatment which has found favour more especially with many American physicians. In cases where there is a personal or family history of rheumatism, salicylate of soda may be given with advantage in the earlier stages of the attack. Antipyrin, in doses gradually increased up to two drachms in the twenty-four hours, has had a reputation in chorea since Walner's paper appeared in 1887. Its chief use is during the acute stage, and even children tolerate it for weeks. Apart from the more usual signs of intolerance of the remedy, the appearance of albumin in the urine must be watched for, and if it occur the use of the drug must be discontinued. Exalgin, analgene, and asaprol have been similarly employed; Moncorvo being prominent among the physicians who have advocated the use of these drugs and of antipyrin in chorea. Physostigma has its advocates, and there are those who have had recourse to the use of curara in certain chronic cases; in view, however, of the dangers which attend its use, the latter drug is best avoided.

Of the sedative class of drugs chloral has been found the most useful; and good results from its use have been recorded, more especially by Sir William Gairdner and Dr. Charlton Bastian. The patient is kept continuously under the influence of the remedy, the object being to obtain arrest of the spasmodic movements by prolonged sleep. A careful watch must be kept on such patients, and the drug suspended in the event of serious cardiac depression or gastric irritation. It is only in the severer and less tractable forms of chorea that this plan of treatment is to be tried, for it must be admitted that while undoubted good appears to result in some cases, in others the natural course of the disease does not appear to be shortened by it; in others again, though the spasms cease or become greatly reduced during the use of the drug, they return with all their former vigour when the treatment is discontinued. Moreover, such patients may be mentally deranged for a variable length of time after the use of the remedy.

The bromides are of little use except in combination with chloral, when they may avert the headache induced by the latter drug, though good results have been credited to them in chorea gravidarum. Sulphonal, paraldehyde, and chlorolose have each been employed in the manner recommended for chloral. Bourneville and Katz allege a rapid cure with bromide of camphor given in capsules of 20 centigrammes. Few are bold enough to adopt Jaccoud's plan of giving large doses of opium to attain the same end in young children; indeed, morphia as a sedative in chorea is less potent than chloral, and may increase any mental excitement which is present.

Hyoscyamin has been employed; and subcutaneous injections of the hydrochlorate of hyoscin have been useful in severe cases. Cimicifuga, cannabis indica, conium, and similar drugs have had their advocates, though most of them are of doubtful value.

Counter-irritations to the spine, freezing the skin, and similar measures, are not to be recommended.

Electrical treatment has not been attended with any success in chorea, though it has been tried in all its forms. In the later stages of the paretic forms of the disease, however, some good may result from the use of mild galvanic currents to the affected limbs. Similarly, massage is of value in the later stages of chronic cases, a time when carefully planned exercises are also of great value in re-educating the patient in the performance of voluntary movements with precision, and in the proper power, rate and adjustment of movements. The patient may be directed to touch a certain point with the finger or toe, to make traction with the limb against various degrees of elastic resistance, or to touch different points with the finger or toe a varying number of times in equal intervals. Besides this the patient may be taught to imitate various simple exercises, and to practise the same before a glass, where defects are more readily detected, and the influence of the will is brought more and more to bear on the movements. In the performance of all these exercises care must be taken not to fatigue the patient. Repose should be encouraged in the intervals between the exercises.

Passive movements also serve a useful purpose; for, in addition to their effect on the muscles, after a movement has been performed in this way several times it can be performed voluntarily with greater vigour and rapidity.

Hydropathic treatment is also of value in chronic cases where a tonic effect is desired, baths and douches being both useful. Sulphur baths also have yielded good results.

CHRONIC PROGRESSIVE CHOREA

SYN.—*Chronic adult chorea ; Hereditary chorea ; Huntington's chorea ; Senile chorea.*

This form of chorea manifests itself in adults after the middle period of life, and has a chronic course which is persistent and progressive. Some authors have regarded "Senile chorea" as distinct from "Hereditary" or "Huntington's chorea," as in the former group of cases there is no hereditary history; others have contended that they are one and the same affection, the negative family history being an insufficient reason for separating maladies which in all other respects are identical. Many of the isolated cases of senile chorea conform so closely in their manifestations to those of the hereditary form, that they cannot reasonably be placed in a different category ; but some of them differ from the hereditary form not only in the absence of any family tendency, but also in their benign course, in the absence of any mental affection, and in their course to recovery, all of which points, as we shall presently see, are foreign to hereditary chorea.

HEREDITARY CHOREA.—Though Huntington, in a paper published in 1872, was the first to give a comprehensive description of the affection which bears his name, there is evidence that the affection was known to American physicians at least thirty years before. Moreover, Huntington's father and grandfather had both been familiar with the disease and its characters during a period extending over seventy-eight years. Since Huntington described the affection, instances of its occurrence in this country, in Germany, and in France have been recorded.

Causation.—The most prominent etiological factors are the liability for several members of the same family to become affected, and the frequency of direct hereditary transmission. Remak instances a family in which nineteen members were affected ; and Lannois one in which there were seventeen such cases ; while the disease has been known to be transmitted through as many as four generations. It does not appear to be more commonly transmitted through one sex than through the other ; and a marked feature in the transmission is that if one generation escape, subsequent ones remain free from the disease ; it does not skip a generation or more and then reappear. In some families the proportion of men affected have been in excess of the women ; but, as a rule, the two sexes are equally liable to the disease. The symptoms usually manifest themselves between the ages of thirty and forty; but instances have been recorded in which the disease began before this, in a case recorded by Hoffmann in early adolescence. No immediate cause can, as a rule, be detected, though in some cases moral emotions have appeared to act in this way. Neither rheumatism, the infectious fevers, nor pregnancy play any part in the etiology of the disease.

Morbid anatomy.—Nothing pathognomonic has been found, though the careful investigations that have been made by several observers—notably by Kronthal and Kalisher, Oppenheim and Hoppe, Dana, Clarke, and Collins—have proved the existence of definite morbid changes. In a good many of the cases the dura mater has been found in a condition of chronic pachymeningitis, with or without hæmatoma; but in one examined by Berkeley there was atrophy of this membrane. Nearly all the cases have presented evidences of a chronic pia arachnitis, with a varying degree of adhesion of the pia mater to the surface of the cerebral cortex. The most common changes met with in the brain itself have been either characteristic of a primary degeneration of the neurons, or of a chronic encephalitis. Macroscopically, atrophy of the convolutions, widening and deepening of the sulci, and compensatory external hydrocephalus have been found. Increased density of the cerebral substance, with thickening of neuroglia, has been described by some, while Collins found a cribriform appearance of the brain in his case. Small foci of softening with vacuolation of nerve-cells has been met with; but the most frequent changes, according to certain observers, consist in disseminated foci of round cells in the cortex and subcortical white matter; a regular diffuse encephalitis, according to Oppenheim and Hoppe. Lannois and Paviot have found similar interstitial infiltration most marked about the large pyramidal cells in the central convolutions; atrophy of the small round cells between the first and second cortical layers was found by Oppenheim and Hoppe, and Menzies found the nerve-cells of all the layers degenerated. In several of the cases examined the walls of the cerebral vessels were thickened. Kronthal and Kalischer concluded that the change in the cerebral cortex consists essentially in disease of the blood-vessels, which not uncommonly leads to proliferation of nuclei, punctate hæmorrhages, and hyperplasia of glia and interstitial tissue, while the nerve elements themselves are but little affected. On the other hand, the findings of Dana, Clarke, and Collins respectively show the essential change to be a degeneration of the ganglion cells of the cerebral cortex; the last-named observer found the large pyramidal and polymorphous cells most affected. In Collins's case the changes, though widespread, were most pronounced in the Rolandic region. The pericellular and perivascular spaces were enlarged, and consecutive and secondary changes were seen in the interstitial tissue and vascular system. Diffuse degeneration of the pyramidal tracts was met with in the pons and medulla by Kronthal and Kalisher, who also found a similar change in the antero-lateral columns of the spinal cord, in which region Oppenheim and Hoppe, and Lannois and Paviot found diffuse sclerosis also. Collins in his patient found slight degeneration of the crossed pyramidal tracts in the spinal cord.

Some of the cases in which morbid changes have been described probably belong to the class of cases of senile chorea in which there is no known heredity; others perhaps should be relegated to the class of athetoid spasms, notably instances in which hæmatoma of the dura or other gross changes have been found; but many of the cases were genuine instances of hereditary chorea.

Pathology.—The pathology of the disease is obscure. The histological changes met with by certain observers point clearly, as we have seen, to a parenchymatous degeneration, in which the neurons suffer primarily, and in which any interstitial change is secondary; but others have found appearances which suggest that a vascular and interstitial change is the basis of the morbid condition. The former of these views has certainly most to recommend it in the present state of our knowledge of the subject; but none of the changes can be regarded as specific. We know little of the nature of the hereditary influence which is so prominent an element in its etiology, though that it depends on some pre-natal factors there can be little question. We are no more fortunate in our knowledge of what determines the manifestations of the disease at the particular time of life when these most commonly first show themselves. According to Dana, and Lannois, and Paviot, the defect is to be found in teratology—a hereditary malformation. The latter observers locate this abnormality in the interstitial tissue, while Greppin believes that certain epithelioid cells remain embryonic, and, later in life, form the starting-point of the disease. Dana regards the pyramidal cells themselves as possessed of a hereditary taint, there being less power of resistance to excitations. Collins, on the other hand, considers that the ganglion cells are genetically wanting in power to survive as long under the same environment as the ordinary ganglion cell.

Symptoms.—The onset is as a rule gradual, though instances of sudden onset on emotion have been met with. The motor disturbances are usually the first to manifest themselves, and may consist either in irregular movements, first noticed in the face or arm, or in the unsteady gait. Symptoms indicative of mental derangement have rarely preceded the motor manifestations of the disease.

The spasmodic movements resemble those of ordinary juvenile chorea in being involuntary, irregular, and without rhythm; but they differ in being much slower, and, in some cases according to Osler, incoördination may be the chief evidence of derangement of the motor processes. In the earlier stages, so slight are the muscular contractions, there may be little or no resulting movement of the limbs; but when the disease is more advanced the spasmodic movements occur both during rest and during some voluntary act; they are increased by emotion, they are lessened by repose. Sometimes they may be arrested voluntarily; but this only results in the earlier stages, when the will retains a limited and temporary influence. Sometimes the involuntary movement is arrested by the substitution of a voluntary effort. The spasmodic movements cannot be arrested voluntarily when the disease is fully manifested; the parts are then in constant agitation, and the movements are suspended only during sleep.

No part is exempt from the spasmodic movements: the ocular muscles have been implicated, causing rolling movements of the globes; grimaces are caused by slow contractions of the facial muscles; contortions of the tongue occur; and in consequence of the spasms the act of deglutition may be difficult. Speech is affected chiefly because of the disordered movements of the tongue. At first it is slow and halting, later the words are pronounced very indistinctly; moreover, the speech may be interrupted by inspiratory or expiratory sounds, the result of laryngeal spasms. The irregular movements of the upper limbs cause characteristic alterations in the handwriting; and as the disease progresses it becomes impossible for the patient to write at all. The attitude and the swaying of the trunk, especially when the patient is walking, resemble closely those of a drunken person. This is very characteristic when fully manifest; the patient may not only sway from side to side, but may lurch more abruptly in one direction, pulling himself up in time before actually falling. Finally, walking becomes altogether impossible, but this result may be delayed for many years.

Muscular power is not diminished until the disease is well advanced. In exceptional cases the muscles are said to waste; but no qualitative alterations in the electrical reactions have been recorded.

There is no defect of general sensibility, and the special senses also escape. The tendon jerks are usually increased. There is no affection of the sphincters.

One of the most striking features of the disease is the progressive mental deterioration. At first the patient may be depressed or irritable, memory fails, and the intellectual faculties become blunted. Associated with these manifestations of mental change is a strong tendency to suicide, which, however, ceases in the phase of complete dementia. There may be periods of excitement during the otherwise progressive course of the dementia, and there may be hallucinations at such times.

The symptoms persist until death, an event which may not occur for even thirty years after the first manifestations of the disease; it is preceded by marked cachexia in association with the dementia.

Therapeutical measures are powerless to alter the inevitably fatal course of the disease.

Senile Chorea.—This form of chorea occurs in old age, or earlier in the prematurely degenerate; it is not so rare in this country as is the hereditary form. It differs from juvenile chorea in that it is in no way related to rheumatism and endocarditis; any cardiac lesions met with being purely of a degenerative kind. From Huntington's chorea it is distinguished by the absence of family proclivity, by the frequent absence of mental change, and by the occasional termination in recovery.

Some difference of opinion, however, exists among authors in respect of these distinctions. Charcot, Huet, and, more recently, Féré, have regarded the chronic adult forms of chorea as differing from chorea minor only in their substratum; others again regard the isolated cases of senile chorea as identical with the hereditary form. Osler, for instance, who is an advocate of the latter view, regards heredity, as in Friedreich's ataxy, as only one and an inconstant feature of the chronic progressive choreas.

Causation.—The manifestations of the disease have been known to begin at all ages between fifty-five and eighty; rarely before this time of life. Men and women are both liable to be attacked, the former in greater proportion than in the juvenile form. A family history of other neurotic affections is sometimes to be obtained. Shock or anxiety has acted as an incidental cause in some cases, but in many no such influence can be traced.

Morbid anatomy and pathology.—Such changes as have been met with after death are similar to those described in hereditary chorea. Here, too, some of the changes may possibly be secondary to the chronic over-action of the nerve elements; indeed, whether primary or secondary, we know nothing of the way in which they are brought about.

Symptoms.—Little need be said of irregular spasmodic movements which resemble those of other kinds of chorea. The upper limbs are usually more affected than the lower, and articulation may be greatly interfered with by the spasms of the muscles of the face and tongue. A certain degree of muscular enfeeblement may be present. Emotion increases the spasms, sleep generally suspends them; they may continue over long periods of years until the patient's death without appearing to shorten life; in exceptional instances, indeed, patients have recovered after a few months or years.

Treatment.—The few patients that have recovered were treated by the usual drugs of recognised value in juvenile chorea, notably arsenic. Sedatives must be judiciously employed to give temporary rest from the incessant movements, especially in cases in which sleep is prevented by them.

Congenital chorea.—There are two different conditions to which the name congenital chorea has been applied: in the one the choreiform movements occur in association with rigidity and exaggeration of the tendon jerks, in the other there is no such rigidity.

Four cases of the flaccid variety have been collected by Ballet, one of which came under his own observation. Males and females are equally liable to be affected, and no circumstances attending the delivery of such children throw any light on the causation. The spasmodic movements begin a few days after birth. In Ballet's case the face and upper limbs were affected first. The spasms are irregular and arrhythmical, of considerable range, are made worse by voluntary movements or emotion, and are suspended in sleep. No cardiac lesions are present.

Though the subjects of this form of congenital chorea are not idiots, they are slow and backward, their intellectual development being below the normal standard. The course of the affection is chronic, and the movements persist in spite of treatment.

This variety is regarded as belonging to the same category as the hereditary chorea of Huntington.

In the cases in which the choreiform movements were associated with rigidity and an exaggerated state of the tendon jerks, labour was difficult and premature, and the children were idiots; there seems little doubt, therefore, that these cases belong to the group of cerebral diplegias, and that the involuntary movements should be regarded as a form of athetosis rather than as a variety of chorea.

ELECTRIC CHOREA.—The name "electric chorea" has been applied to various and widely different conditions, but is best reserved for an affection known also as "Dubini's disease," as this physician first described the condition, in 1846.

The other conditions to which the name "electric chorea" has been applied are certain cases of ordinary chorea, in which the spasmodic movements are more sudden than usual; cases of hysterical chorea with shock-like movements; and an affection which resembles hysterical chorea, and in which rhythmical spasms involve the neck muscles more especially. This "electric chorea" of Bergeron is a benign affection which rapidly ends in recovery.

Causation.—"Dubini's disease" occurs most commonly in Lombardy and neighbouring parts of Italy, and appears to be due to an acute infection. Its occurrence in certain localities has suggested that it is related in some way to the malarial poison; but the affection is rare even in those parts, and in many other malarial districts it is unknown. Males and females are both liable to be attacked, and it occurs at all ages.

Morbid anatomy.—No constant changes have been met with in the central nervous system, but the meninges and spinal cord have frequently been found congested.

Pathology.—There is much in the clinical manifestations of the disease to suggest an acute infection. That the toxic agent first attacks the neurons of the cerebral cortex is suggested by the frequent unilateral commencement of the spasms, and by the occurrence of convulsions; but that the neurons of the anterior horns of the spinal cord eventually become affected seems certain from the muscular atrophy which supervenes; however we have no anatomical evidence in support of either conjecture.

Symptoms.—The disease is ushered in by pains in the neck and back; the temperature is raised in proportion to the degree of acuteness of the case, and may be raised throughout the course of the affection. Spasmodic movements occur as one of the earliest manifestations; one of the upper limbs is affected first, the movements beginning either in the upper part of the arm or in the fingers. Next, the spasms most commonly affect the muscles of the leg of the same side, before becoming generalised. The muscular contractions differ from those which occur in ordinary chorea by being sudden and shock-like, exactly like those evoked by momentary electrical excitations. In addition to these movements epileptiform convulsions occur in a good many of the cases, and are sometimes restricted to one side of the body. Palsies may follow such attacks; but, apart from this, in patients who survive for some months, the limbs become feeble in the order in which they were affected by the shock-like spasms, the paresis in the end becoming general. The muscles waste and no longer respond to faradism.

The disease pursues a progressive course to a fatal termination, which is usually postponed for several months, but may take place after but a few days' illness. Death may follow an epileptiform convulsion, or there may be a gradually deepening coma.

Treatment does not appear to be capable of altering the progressive course of the disease, or of averting death.

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THE TICS

History.—Thanks to the French school, especially to Charcot and his pupils Gilles de la Tourette and Guinon, more or less order has been substituted for chaos in the large group of affections of the nervous system characterised by muscular spasms. To the Salpêtrière school is mainly due the credit of separating the various forms of tic from the choreas, and that it is difficult to over-estimate the importance of such a discrimination may be judged from the words of Charcot on this subject: "Entre le tic et la chorée il y a un abîme"; and again, "Sans doute, nosographiquement, les tics et la chorée représentent bien, comme je vous l'ai dit, deux affections radicalement distinctes."

The word "tic" means a "jerk" or "twitch," and admirably describes the form of motor disturbance met with in the group of cases to which the French have applied the term. This nomenclature is, however, not without its drawbacks, for, both in this country and in America, the term "tic" has been applied to the condition otherwise known as facial spasm ("tic non-douloureux"), or to facial neuralgia ("tic douloureux"); while the conditions to which the French now apply the term are what we have been in the habit of designating "habit-spasm," "habit-chorea," and so on.

The classification of the tics now in vogue among French writers has been well set forth by Noir, and is as follows:—

1. *Simple tic.*—This is essentially the condition to which the term "habit-spasm" has been applied by Sir William Gowers, and which Dr. Weir Mitchell designates "habit-chorea." The latter name is singularly inappropriate, as the movements of this form of tic do not in the least resemble the movements of chorea; and the former appellation, while having more to justify it in that some cases of the affection do appear to arise out of a trick or habit, is by no means strictly applicable to all cases of simple tic, seeing that many can in no way be ascribed to any trick or habit which has subsequently become automatic.

2. *Convulsive tic* or *Maladie des tics convulsifs* is an affection in which, in addition to the motor disturbances which characterise the simple tic or so-called habit spasm, explosive utterances and imperative ideas form a prominent part of the clinical picture. In France this variety of tic is commonly spoken of as Gilles de la Tourette's disease, as this physician has made it the subject of much careful study. In some of its aspects this condition is so similar to the malady known in America as the jumping disease of Maine, among the Malays as latah (*vide* art. "Latah" in vol. viii.), and in Siberia as myriachit, that in all probability the affections belong to the same category, or are even identical.

3. *Co-ordinated tics* or *Tics coordonnées* differ from the simple tics in the character of the movement, which, instead of being a simple jerk or twitch, consists in some complex movement in every way similar to those which we execute in the ordinary routine of life; the only difference being that the particular act is repeated time after time without obvious reason. To Noir is due the credit of an admirable description of this variety of tic.

4. *Psychical tic* or *Tic psychique* is essentially of the same nature as a motor tic, the spasmodic movement being replaced by its psychical equivalent—an imperative idea; or, as Charcot expressed it, "Il y a des tics dans la pensée comme dans le corps."

SIMPLE TIC.—Syn.: "*Habit-spasm*," "*Habit-chorea*."—In simple tic spasmodic movements occur which seem voluntary, but are performed with lightning-like rapidity, and are beyond the control of the will. The condition has been regarded as a form of chorea by Weir Mitchell, whose view is based on the fact that children are usually affected; that it is associated, as a rule, with a lowered state of health, irritability, and nervousness; that sometimes habit-spasm lapses into chorea; and, lastly, that the remedies which are the most effectual in treatment of this condition are those which have been found of greatest value in the treatment of chorea.

Plausible as these arguments may appear, and while, indeed, it must be fully admitted that in some instances it may be difficult to be certain whether the case be one of chorea minor or simple tic, there can be no question that the two conditions are entirely distinct, and that between them there is an immeasurable space. Most modern writers who have given attention to the subject have come to regard the two affections as distinct, and even Sinkler, who supports Weir Mitchell's view that habit-spasm is a form of chorea, has to admit that there are cases of the kind to which the term habit-spasm is correctly applied, and which are to be classed with the different forms of tic.

Causation.—*Heredity.*—Direct heredity can rarely be traced, though Sir William Gowers mentions a case in which a father, the subject of habit-spasm of the face, with which he was affected throughout his life, had two children who became similarly affected; but here imitation may have been operative. Sinkler refers to a case in a boy, two of whose aunts had suffered from a form of habit-spasm. Commonly, however, other neuroses are present in parents and relations, and more than one child in a family may be affected.

Sex.—Females were formerly supposed to be more commonly affected than males, but sex appears to exert no influence on the frequency of the affection.

Age.—The malady is chiefly met with in childhood, most commonly in the second hemi-decade; but a large number of cases also occur during the third hemi-decade, and nearly 80 per cent of all cases begin between the ages of five and fifteen years. Instances of its beginning at the age of one year have been recorded; on the other hand, it may be postponed until adult life, though Sinkler met with no case beginning after the age of thirty-seven.

Habit.—In many cases the condition originates in some trick or habit so often repeated that it ultimately gets beyond the voluntary control of the individual. In some cases the trick is acquired without obvious reason, while in others the spasm originates in some cause inducing either voluntary or reflex movements. Such spasmodic movements may, however, persist after the exciting cause has been removed; thus a conjunctivitis may be responsible for blinking of the lids, which movements persist after the conjunctival affection has ceased.

Imitation.—Probably some cases owe their origin to imitation, not necessarily a direct imitation, but rather an irresistible tendency to give vent to pent-up feelings of unrest engendered by the witnessing of similar movements in another.

Lowered general health.—A state of lowered health, variously induced, as by previous acute illness, overwork, growing too fast, and the like, commonly precedes the onset of the affection; anæmia, again, may play a part, though its occurrence does not seem to be so common as in chorea. On the other hand, it may be that without any obvious impairment of general health, influences have been at work which have had a depressing effect on the nervous system, such as shock from an injury or fright, mental overwork, and so on. As in chorea, so in tic, there are cases which appear to be school-made.

Reflex irritation.—Conjunctivitis causing blepharospasm has already been mentioned. Obstructive diseases of the nose, including adenoids and the like, are commonly present, and occasion sniffing movements of the face, which, voluntary at first, subsequently become automatic. Or the source of irritation may be remote from the seat of spasm; no more potent cause of this kind is to be found in boys than masturbation.

Optical defects.—Though deserving of separate consideration, there is no essential difference between these conditions and those that have just been considered. Of forty-nine cases of Sinkler's series, which were examined by De Schweinitz and Thomson, errors of refraction were found in forty-one of the patients; in two there was conjunctivitis, and in six there were defects in ocular balance.

Season.—No relationship between simple tic and season is known; the cases appear to be distributed in about equal proportions over the different periods of the year.

Relation to Sydenham's chorea.—Cases of simple tic have followed chorea, and the former condition has been said to lapse into the latter. In Sinkler's series of 113 cases simple tic and Sydenham's chorea coexisted in two patients, while in nine the latter condition preceded the former.

Symptoms.—The spasmodic movements in tic are characterised by the suddenness and lightning-like rapidity with which they are executed. The form which the movement takes varies not only in different persons, but also in the same person, so that two or more kinds of movements may be prone to occur, now one and then another manifesting itself; or again a certain movement may exist alone for a considerable time, when another is added, or it replaces that which previously existed. The interval which separates the succeeding movements varies considerably; usually a few minutes elapse, but at other times the movements follow each other in such quick succession as to become almost incessant.

The distribution of the spasms, also, is subject to considerable variation; but the face is the part most commonly affected, and here blinking movements of the eyelids are more frequent than any other form of spasm; another common movement is that of alternate elevation and lowering of the eyebrows. Contraction of the zygomatic muscles, with drawing of the angle of the mouth to one side and then to the other, is also of frequent occurrence. Sudden jerkings of the head are common; the head is in consequence displaced backwards, forwards, or laterally, or the movement may be one of rotation. The platysma is not uncommonly involved in the spasm, either alone or with other muscles of the neck. The upper limbs are also frequently affected; the most usual movement is shrugging of the shoulders, but, in other instances, various other rapid movements may be met with, such as flexion or extension of different joints, or rotation of the whole limb. Spasmodic movements of the lower limbs are very much less commonly observed, though sudden extension with stamping of the feet and the like may occur; or sudden flexion of the leg or thigh at irregular intervals during progression, constituting what has been spoken of as "Springhalt tic."

Affection of the muscles of respiration is common, and not only is the rhythm of respiration thus deranged, but various grunts and other similar sounds are emitted; these may be laryngeal in character and of varying intensity, or like a sob or sniff. Any of these sounds may be frequently repeated, and may be associated with some disordered movement of the face, trunk, or limbs. Another troublesome symptom is cough, which may be hacking or very loud and oft repeated.

According to Dr. Osler, cases in which there is a sudden start, due to a general spasm which passes through the muscles of the trunk and limbs with electric-like rapidity, may be conveniently included as a generalised form of simple tic with the cases we are now considering. The spasm is almost instantaneous, passing off with great rapidity, to be repeated in similar fashion from time to time. It is to this variety of tic that Henoch has unfortunately applied the name electric chorea. The name is unfortunate for two reasons: the condition is not a form of chorea at all, but belongs to the family of tics; moreover, it has been given to a totally distinct affection met with in Lombardy, and known as Dubini's disease (p. 864).

The movements in habit spasm are increased by any excitement, especially under observation, though the effort to concentrate attention during medical examination may lessen the frequency of the spasms; an inhibition which never occurs in chorea. An effort of the will may or may not control the spasms, and sometimes the patient is able to resist the impulse to perform some act for a time; though in the end he is obliged to yield, as it were to an imperative mandate. The movements always cease during sleep.

The spasms are usually much more limited in range than is common in ordinary chorea; but when they continue for a long time they may spread, so as to involve several groups of muscles. As a rule the habit movements cease after a time; or again they sometimes persist to be a source of great annoyance in adult life.

Prognosis.—Among the chief of the several factors which influence prognosis is duration; the longer the tic has persisted the more difficult it becomes to alter the tendency to involuntary action which the motor neurons have acquired, particularly if the patient be an adult. On the other hand, if attended to early, more especially if the patient be a child, the spasms may be corrected; and the cure thus brought about may be permanent. Another important element in prognosis is the age at which the manifestations first occur, for, according to Sir William Gowers, when it begins late in life the affection is generally permanent. It must be remembered further that not only may the condition be permanent in any given case, but also that the range of its influence may be so increased that ultimately many more groups of muscles may become affected than those originally concerned.

Treatment.—It is of primary importance that every case of the kind should be carefully investigated with a view to ascertain any cause for the condition, and to remove or counteract its influence. Yet, even when detected, the removal of the cause will not, as a rule, alone suffice to restore tranquillity to the motor neurons at fault, sufficient as it may have been to induce the morbid state in the first instance. A vicious habit of the nerve-centres is easily acquired, but difficult to break, more especially when of long duration; so that we have not only to remove the cause, but also to adopt measures to restore stability to the motor neurons. Thus it is not only necessary to treat the condition of the nose, or correct the error of refraction, as the case may be, but also to employ measures to build up the system generally, and thus secondarily to attempt to improve the nutrition of the motor neurons.

In the majority of cases, indeed, the general health is impaired, and there may be, as we have already seen, a certain degree of anæmia. Improved hygienic surroundings, fresh air, or change of air, together with good food and tonics, are called for. All chalybeates are of value, but no drug exerts so great an influence over the disorder as arsenic, given in gradually increasing doses until its toxic effects become manifest. Cases which do not yield to the drug when administered by the mouth may, nevertheless, do so when it is given subcutaneously, as was first pointed out a good many years ago by Weir Mitchell. Strychnine is also of advantage, as a substitute for the arsenic, from time to time. In the more aggravated forms of the affection a sedative line of medicinal treatment may be called for, in which case some form of bromide should be given contemporaneously with nerve tonics.

The drugs that proved of most advantage in Hammond's hands are conium and atropine, and he has recorded some striking instances of cessation of the tic under their influence. He considers that the value of these remedies is enhanced by combining them with moderate doses of a bromide. In the case of atropine, Hammond gives $\frac{1}{100}$th of a grain as an initial dose, and gradually increases it up to $\frac{1}{10}$th of a grain; in the case of conium he finds the fluid extract or conine equally efficacious, and, beginning with five minims of the former, he increases the dose by one or two minims a day until the tic ceases, or the toxic effects of the drug become manifest; in the latter case, if the tic still persist, he reverts to the original dose, and gradually increases the amount again as in the first instance.

When the limbs are affected gymnastics are of advantage, as in true chorea; and massage is of like benefit.

As little notice as possible should be taken of the spasms; but the patient should be encouraged to control them by an effort of the will. In children the promise of reward may be thus effectual; but we earnestly deprecate any actual or threatened punishment if such voluntary effort fail, or indeed be not steadily made.

Rest in bed is highly beneficial in some obstinate cases, and the patient should never be allowed to work, or to follow exciting pastimes, his life, indeed, should be as quiet as possible, short of monotony.

CONVULSIVE TICS.—Syn.: "*Maladie des tics convulsifs*". "*Gilles de la Tourette's disease.*"—We have no very satisfactory name for this affection; for neither "convulsive tic" nor "maladie des tics convulsifs" implies much more than occurs in simple tic; whereas, as we have already noted, in the variety of tic that we are about to consider, explosive utterances and psychical phenomena are added to the spasmodic movements, and form a striking part of the clinical picture.

The affection is a neurosis characterised by sudden lightning-like muscular contractions, which are repeated without the slightest rhythm, and may be either limited or generalised. In addition to this the victims are liable to involuntary explosive utterances, in which case obscene words are sometimes emitted (coprolalia), or there is an irresistible impulse to repeat words or sounds (echolalia), or to imitate gestures (echokinesis). Psychical troubles also occur in which obsessions and imperative ideas play a prominent part.

To the writings of Charcot, Gilles de la Tourette, and Guinon we are indebted for a clear account of this affection; and neurology owes much to the school at the Salpêtrière for having separated this form of tic from the hysterias, and from a large class of indefinite conditions in which spasmodic movements play the chief part.

Although Charcot inspired his pupils to do the excellent work which has placed this affection on so secure a basis as an independent condition, differing in effect from other neuroses with which it is liable to be confounded, in fairness to Trousseau it must be stated that he clearly recognised the affection and indicated its chief features.

The Salpêtrière school and various other writers have regarded the muscular tics as dominant in the early part of the clinical history of these cases, and echolalia, coprolalia, echokinesis, and the like, as subsequent accretions. Other writers, however, accord the first place in the clinical picture to the psychical tics which may occur exclusively at the beginning of the malady. Tokarski, by attempting to separate the two groups, has gone a step farther, and would reserve the name "maladie des tics convulsifs" for the former class, and "myriachit" for the latter. The term "myriachit," however, as I have already said, has been long in use in Siberia to denote an affection which is probably identical with the variety of tic that we are now considering; and it is questionable how far a subdivision such as that suggested by Tokarski is warranted in the present state of our knowledge of this subject, though Noir upholds the distinction.

Causation.—Heredity plays a most important part in the causation of this affection, and may be either direct—some of the ancestors having been the subjects of tic—or indirect, various other neuroses or degenerative conditions of the nervous system having been prominent among them. So, too, it would seem that a drunken parent may beget offspring prone to tic. This factor appeared to be operative in a case recorded by Chabbert, in which a girl born four or five years after the father had taken to drink was ill-developed and the subject of tic, while her brothers and sisters, born prior to this, were in every way perfectly healthy. Indeed, according to Guinon, the maladie des tics is always an expression of hereditary degeneration, however difficult it may be to secure evidence of it.

Whether it be possible for a nervous system originally normal to acquire the affection or not, congenital defect is proved by the frequency of malformations and other stigmata, congenital or acquired, in the subjects of convulsive tic. Diseases occurring in infancy may, however account for some cases.

Males are a little more frequently the subjects of the affection than females; and the first manifestations of the malady are usually observed in childhood, most cases beginning during the second and third hemidecades; though there are instances on record in which the subjects remained free up to adult life. In some of these, at least, it is probable that more searching inquiry would have revealed some rudiments of the disorder at an earlier age.

The outbreak is usually evoked by an accidental cause, especially by mental shock; but physical shocks and injuries are also operative. It does not follow that the spasmodic movements affect the injured part; on the contrary, these may entirely escape, as in two cases recorded by Chabbert, in which a mother and son were both the subjects of localised tic, and in neither of them did the injured part participate in the spasmodic movements. Other immediate causes which, however, play a much less important part in the ætiology of the affection are infections and intoxications. Moreover, convulsive tic is engendered in some cases by imitation. Guinon mentions the case of a child who saw a person in an epileptic fit in the street; he was frightened, and was attacked with the contortions of the mouth which he had seen in the epileptic. Another child, in perfect health, went to a café concert, where he saw a performer make grotesque grimaces to amuse the audience, and in a few days he reproduced the grimaces in spite of himself. The door once opened, such subjects manifest in the course of time all the typical manifestations which make up the complete picture of the maladie des tics.

Symptoms.—As already observed, four groups of symptoms characterise the clinical picture of the affection—spasmodic movements, uncontrollable utterances, an impulsive tendency to imitate by acts or words, and a peculiar mental state in which obsessions and imperative ideas play an important part.

The spasmodic movements are, as a rule, sudden and lightning-like; though it is said by Chabbert that in exceptional cases they may be quite slow and deliberate; in any case they are repeated in the most irregular way possible, without the slightest rhythm. The resulting movement may have no resemblance to any ordinary voluntary act, or, on the other hand, movements of defence, gestures of contempt or defiance, and the like, may be forthcoming.

The muscles of the face and neck are especially prone to participate in the spasms, while the limbs are less liable to be affected, the lower extremities escaping more often than the upper, but no part of the body is exempt, and in some cases there is scarcely a muscle that does not participate in the spasms at one time or another; though, according to Kahler, generalised spasm is very exceptional.

In the face the spasms result in all sorts of grimaces and jerky movements; the eyes are rolled in this direction or in that, the forehead wrinkled, as in frowning or in the expression of surprise; now the eyes are too widely opened, at the next moment they are closed. The sniffing movement, so common in simple tic, is frequently present, and various contortions occur about the lower part of the face and mouth, the latter being at one time opened, at another time closed; or mastication movements, perhaps with grinding of the teeth, may be seen. A variety of movements of the tongue, including protrusion and retraction, may occur.

The head is jerked in every conceivable direction by the spasmodic action of the neck muscles; not uncommonly it is suddenly retracted, and a quick lateral movement is added; or the chin may be brought forcibly in contact with the chest; so, too, various lateral and rotatory movements of the head occur from time to time, and the platysma is frequently seen in action.

One of the most common movements of the upper limbs is that of shrugging of the shoulders, which may be bilateral, or now one and then the other shoulder is suddenly raised and then let fall. So, too, other movements of the shoulders occur, as for instance a twist as if to get rid of some discomfort, or sudden forward or backward movements of this part. The whole arm may be raised, or any of its component segments may be moved in this or that direction, the fingers participating in the abnormal movements, all of which are executed in the same brusque, jerky fashion.

The muscles of the trunk may take part in the spasms, when various movements of propulsion, retropulsion, or lateral oscillations of the body are seen; and these, alone, or in conjunction with any spasmodic movements in the lower limbs, may interfere with progression. Isolated movements of the trunk muscles are, however, uncommon, though the salaam movements thus occur in some cases.

Although the lower limbs are more commonly exempt than the upper, yet, when affected, the spasms may be quite as severe there as in any other part of the body. A stamping movement of the foot is not uncommon, and the limb may be suddenly extended as in the act of kicking.

Sudden flexion of the leg and thigh may result in a spring-halt movement; or the patient may suddenly stoop, or jump, and so on. These various anomalies give rise to the most grotesque modes of progression: for instance, the ordinary manner of walking may be suddenly interrupted by a few dancing steps, or by a jump on both feet, or a hop on one, by a sudden kick forward of one or other limb, and the like. In a case recorded by Gilles de la Tourette, the patient ran rapidly for a short distance, then touched his knee and performed the most extraordinary movements with his arm.

The spasmodic movements occur in attacks, and are not, more or less continuous, as is the case in chorea; in each attack several groups of muscles may be in action simultaneously; or the contraction of one group may follow that of another, in which case there is a curious tendency to preserve the same order in successive attacks. As I have said, the tic may be limited in distribution, and the limitation may be to one side of the body,—an event which, however, rarely obtains in the case of the face in which the affection is usually bilateral.

The patient is fully alive to the fact that the movements occur; indeed, he may be painfully conscious of the ridiculous grimaces and contortions which he is making, nevertheless he has practically no voluntary control over the movements. A voluntary movement of some kind is a surer device by which to escape the spasmodic movement than a mere effort of the will, although a very powerful effort of the will may be successful; but so great is the effort required that it is most fatiguing to the patient, and may make him feel so ill that the spasmodic movement is preferable to the malaise. The devices adopted by choreics to mask the involuntary movements by the performance of some voluntary one in quick succession to that of involuntary initiation is seen in the subjects of tic also; but with so little success, as a rule, that the victims of tic are thankful for solitude as the only way of escaping observation.

The movements always cease during sleep, though Féré mentions a case in which attacks sometimes occurred during sleep, in his opinion probably as the result of dreams. Apart from actual sleep there is much less tendency for the attacks to occur during repose and in solitude; the quieter the patient the more likely he is to be free from spasm. It is an interesting fact that, as in epilepsy and other spasmodic affections, the spasms may be entirely in abeyance during the course of any febrile disorder. On the other hand, observation, excitement, or emotion of any kind induce the attacks. Disorders of digestion and the like may increase the tendency to attacks, which in women may be much more frequent at the monthly period than at other times.

Apart from any interference by the spasm, voluntary movement is not affected; such patients may be able to perform acts requiring the greatest delicacy of co-ordination, such as playing the piano, writing, and the like, without the least evidence of any defect; indeed Guinon refers to one patient who could juggle with knives without a slip or mishap. The absence of any alteration in the character of the handwriting may prove an especially valuable point of distinction between this affection and certain of the other motor neuroses in which the handwriting is notably altered. There may, however, be some difficulty in writing if tic of a severe type affect the arm.

Although the spasmodic movements are usually the first phenomena of the malady, yet a tendency to explosive utterance may be added; or the initial phenomena of the affection may be of this character, and it may remain at any rate for a long time the sole symptom.

The *exclamatory tic* consists in the habitual emission of some irrelevant word, or parts of words, or sentences, or again of some inarticulate sound, resembling, it may be, that of some animal. In some cases obscene or blasphemous words are ejaculated (coprolalia). This is rather common, and is most embarrassing to the unfortunate patients, as, despite all their efforts, such expressions may be emitted most inopportunely. Coprolalia may be manifested in two ways, both of which may occur in the same patient: the words are either spoken in a loud voice, in which case muscular shocks accompany their emission, or they are uttered in a low voice, and are then unaccompanied by muscular spasms. The spasmodic utterance may be single, or be repeated several times in succession. In a case recorded by Gilles de la Tourette, a youth thus afflicted received a sound thrashing from some other boys who thought that the ejaculation "*couillon*" was directed at them.

Another of these impulses is to repeat like an echo (echolalia).

Words or names may be repeated thus, or the cries of animals and the like; or again, the words echoed may be those of the patient himself.

The echo is usually the end of a sentence, especially of the last word. Various peculiarities are, however, met with in different cases. A word *unfamiliar to the patient or the name of a person is especially apt to impress itself in this way; in any case the word or* phrase is echoed time after time.

Again, movements or gestures may be reproduced (echokinesis). The mimicry may be of a facial grimace, or of some movement of the limbs or trunk; and this feature of the malady it is which bears the greatest resemblance to the tricks of the jumpers of Maine, to the "latah" or "latak" of the Malays (*vide* vol. viii.), and to the "myriachit" of Siberia (Russian "miriatchit" = "playing the fool"). The subjects of this malady are irresistibly impelled to imitate some sound, grimace, or act, although by so doing they may cause pain to others, or bring dire anger upon themselves. Thus in the variety of tic now under consideration—for whatever the form of the uncontrollable impulse, whether the ejaculation of words or sounds, the echo or the mimicry—there is the same powerlessness in the will to prevent it. The impulse may be circumvented by various dodges, but generally with little success; thus voluntary speech may ward off an explosive utterance; but too often some ungainly sound, or an irrelevant word, suddenly interrupts the sentence, or an intended word of welcome to a visitor may be replaced by one that is highly uncomplimentary.

The mental peculiarities, which may form part of the clinical picture, are most remarkable, and belong to the class of imperative ideas—the "idée fixe" or "obsession mentales" of the French. As with the motor tics so ideas may become spasmodic, and, though aware of the mechanical repetition, he cannot help it, and these ideas may lead to importunate, impulsive acts, either meaningless and absurd or even injurious. Sometimes a physical basis can be found for the fixed ideas and automatic acts, but by no means in all cases.

A common form is that of an agonising search for a word or name which when found is repeated time after time; or some word may incessantly obtrude itself on the patient even until he is compelled to vocalise it; the victim may loathe the word or dread it, and may attempt to evade it, but without success. The feature of some cases is an irresistible impulse to count a certain number, or tap a given number of times before some act, or a tendency to touch certain objects.

The condition known as *insanity of doubt* is an abiding uncertainty whether a particular act has been executed correctly or has been forgotten; or there may be an uncontrollable desire to question or analyse the most ordinary acts of everyday life.

We meet with one or other of the various 'phobias—notably agoraphobia, or the dread of walking in open spaces; acrophobia, or the fear of heights; and mysophobia, when the subject is afraid to touch certain objects (*vide* art. "Neurasthenia," vol. viii.)

When these various phenomena are associated in a given case they usually present themselves in definite order; most commonly the spasmodic movements occur first, and are followed by exclamations, coprolalia, echolalia, echokinesis, and fixed ideas—in the order named. It is more common, however, to meet with convulsive tics alone, and, as I have said, the spasmodic exclamations may occur alone for a long time. Echolalia, coprolalia, echokinesis, and the imperative ideas may all be present in the same patient; but more commonly only one or other of these symptoms is met with, or at any rate various combinations of two or three of them.

These fixed ideas obviously border upon insanity; moreover marked loss of will-power is evident in these patients and a loss of the power of attention. They are unable to sustain a conversation coherently, mental application becomes practically impossible; judgment is worthless, and memory suffers, even to the point of inability to read.

It must be recognised clearly that, although convulsive tic and hysteria are totally different affections, there is no reason, as Guinon has insisted, why the two neuroses should not coexist, just as epilepsy and hysteria may be associated in hystero-epilepsy. Not only is the combination possible, but cases are actually met with in which convulsive tic and hysteria are found so interdependent that the presence of manifestations obviously hysterical, or the stigmata of this neurosis, does not exclude the coexistence of spasmodic tic.

Diagnosis.—Difficulties in diagnosis depend largely on the stage of the affection, for when fully established, so that besides muscular spasms the patient emits inarticulate sounds or articulated words, or is afflicted with coprolalia and echolalia, there can be no difficulty in diagnosis; no other malady gives rise to this group of symptoms.

For a long time the majority of these affections were classed as varieties of chorea; but a more detailed study of the characters of the movements in the different classes of cases, and of the symptoms of this or that variety, has led to their proper nosological order. Much confusion, however, still exists, and cases which belong properly to the group of tics are regarded by some as varieties of chorea.

In *Sydenham's chorea* the movements are slower and, as it were, more rounded off, and are not so brusque as in tic; moreover, they are quite incoördinate, and voluntary movements are greatly interfered with, even where no actual loss of motor power is to be detected. The irregular movements do not occur in attacks with periods of absolute calm between them, as in tic; and it is quite exceptional in chorea to see the movements of ordinary life. Volition exercises less control over the movements of chorea than obtains in the case of tic. Again, chorea tends to recovery, however slowly, while the tic is, as a rule, incurable; on this difference we may have to depend for diagnosis in some cases, as for instance when a chorea is not seen until a late stage when a few facial grimaces alone persist. For although it may be impossible from the history of former movements to decide whether the case be one of tic or chorea, yet the latter ends in cure, while no such result obtains in convulsive tic. Certain laryngeal sounds and grunts may result from the spasmodic muscular contractions in chorea, but they are never articulated words; and coprolalia, echolalia, and echokinesis do not occur. Furthermore, although mental defects occur in chorea they are quite different from those of tic; they are true varieties of insanity with derangement of intellect, while in tic the intellect is preserved, although will-power is diminished or abolished, and the patient falls under the sway of fixed ideas.

There ought to be no possibility of confounding tic with the two conditions which have been respectively described as "*Electric chorea*" (Dubini's disease), and *Bergeron's disease*. In the former of these affections the involuntary spasms are accompanied by convulsive attacks which leave a certain degree of paralysis in their wake, and nearly always end fatally, it may be in a few days or after some months (*vide* p. 864). Bergeron's disease, on the other hand, is a benign affection in which the spasm in the face may be arrested by pressure on the facial nerve; indeed the whole of the manifestations of the affection may be dispelled by an efficient emetic.

Nor can there be much likelihood of confounding tic with *chorea major*, which is not a form of chorea at all, but a hysterical manifestation in which the attacks of involuntary movements last a long time, and do not in the slightest resemble the attacks in tic. Automatic or habitual movements of ordinary life are reproduced, patients may dance (chorée saltatoire), imitate the movements of a smith at his anvil (chorée malléatoire), and the like. Moreover, such attacks may be evoked by an emotion, by pulling a limb, by percussion of a tendon, and so on. Such patients either present hysterical stigmata,—such as concentric limitation of the fields of vision, or cutaneous anæsthesia, or a history of hysterical manifestations is to be obtained.

It is rare for *hysteria* to reproduce the whole of the manifestations that make up the complete picture of the maladie des tics; we may meet only with the movements which simulate those of tic, while at another time involuntary vocal phenomena may be present. The laryngeal sounds in hysteria may be grouped around an expiratory spasm (cough), or an inspiratory spasm (hiccough). Such phenomena occur quite suddenly and disappear quite as unexpectedly after having, it may be, resisted all sorts of treatment. The patients are quite indifferent to their condition, and may cough away all day without showing the slightest signs of fatigue. When, indeed, tic is simulated by hysteria, by its spasms only, the movements may be identical. In all doubtful cases search must be made for the hysterical stigmata; but of course a hysterical subject may become affected with maladie des tics, when, according to Guinon, the evolution of the movements alone can indicate the diagnosis; if they cease the case is one of hysteria, if they persistently resist all therapeutic measures then true maladie des tics has been added to the hysteria.

A condition allied to tic, and which may be confounded with it, is *paramyoclonus multiplex* (p. 888). However, the phenomena of imitation are absent, as are spasmodic utterances and mental troubles. Moreover, while tic attacks the face, this part, as a rule, escapes in paramyoclonus: indeed the lower limbs, which are least commonly affected in tic, are among the most usual seats of the muscular contractions of paramyoclonus. Again, the spasms are much more simple, picking out individual muscles or parts of muscles, and usually symmetrical muscles; they never reproduce the gestures or movements of ordinary life. The contractions are isolated, or follow each other in quick succession, and do not constitute attacks, as in tic; furthermore, they may be evoked by cutaneous stimulation, percussion of a tendon, and so forth.

It is conceivable that a tic of limited distribution might be mistaken for a *localised spasm* of a totally different kind. But such localised spasms are uniform, they are limited to a single muscle, to a group of muscles supplied by one nerve, or by nerves so closely related in origin as to be involved in the same lesion; and there is not the multiplicity and difference of movement which make up attacks of tic.

The condition which Létulle has designated co-ordinated tics (*tics coordonnées*) may be attended with movements which resemble the impulsive acts of the psychical tics; but such movements are much more under the influence of the will. They can commonly be traced to some habit, and may not amount to a disease; moreover, some of them differ from convulsive tic in that by force of habit the movements are unconsciously performed; spasmodic utterances and psychical disturbances again form no part of the clinical picture. The subject of certain varieties of co-ordinated tic can arrest the movements by a voluntary effort (p. 883), and such arrest is not accompanied by the distress and subsequent exacerbation of the movements which follow temporary suspension of the movement in the maladie des tics (p. 877); moreover, in some cases, habitual control of the movements in this way may bring about a complete cure.

Facial and other contortions sometimes originate in some slightly painful condition, especially in children in whom ulceration of the mouth or lips, for instance, may set up grimaces. Search should be made for any cause of this kind, either present or past, for under suitable discipline the one form of movement ceases; if in spite of all such control it persist, it is probably a true tic.

Of organic affections of the nervous system, characterised by involuntary movements, *athetosis* and *post hemiplegic chorea* only need be considered, neither of which indeed is likely to cause much difficulty. In the case of athetosis the position of the hand, its continual instability, and the impossibility of its retaining any object in its grasp, are sufficient to distinguish the affection from tic; and many other points of distinction might be adduced. So too, in regard to post hemiplegic chorea, the limb affected by the motor disorder atrophies, and the tendon jerks become exaggerated. Moreover, in neither of these conditions are there any of the symptoms of maladie des tics other than the motor phenomena; for, though mental deterioration may occur, it takes some form of idiocy, never the characters of the fixed ideas.

Prognosis.—*Once established the malady usually becomes progressively worse, continuously it may be, or with intervals during which it appears to be stationary, to improve, or even to become arrested*; such intervals being followed in turn by periods of further increase in the manifestations of the malady. The affection has, no doubt, very exceptionally ended in recovery; yet even if so favourable a result be obtained, it must be remembered that it is peculiarly prone to relapses. In formulating a prognosis it must always be remembered that psychical disturbances may occur, if they are not already present; and that they may end in insanity. Psychical disturbances further influence prognosis in another important way, for so long as the tic manifests itself only by motor and verbal explosions, the duration of life may not be materially curtailed, if at all. It is otherwise, however, when obsessions and impulses harrow the patient, more especially if he struggle to resist them; for of necessity they gain the ascendency, and exhausting himself by his efforts at resistance, he may in despair seek release in suicide.

We must always be mindful of the fact that although in simple tics, such as those limited to the face, the prognosis is relatively good in that such patients may reach adult life without further change, yet a violent emotion, a heated controversy, and the like, may induce a crisis in which new phenomena appear and do not subsequently disappear. When the affection reaches its extreme degree the patient is unable to follow any occupation, or even to live an ordinary life with comfort.

Treatment.—What I have already said as to the incurability of the affection prepares us for the utter impotence of therapeutic measures in nearly all these cases. Drugs of a sedative class are useless; they can do no more than suspend the spasms temporarily while the patient is under their influence; nothing approaching a permanent cure attends such measures. Various tonics are serviceable in a general way, for all measures which are calculated to build up the system, and at the same time are not too stimulating, should be employed; the various preparations of iron are especially useful. Change of circumstances is indispensable in the treatment of these cases, and isolation is of still greater service; of like importance is strict moral and physical discipline, and encouragement in those healthy pursuits or hobbies which prevent introspection. An intellectual patient may divert his mind with study; in other cases some mechanical occupation must be followed. Useful adjuncts in treatment are massage, passive movements, gymnastics, hydrotherapeutic measures, and the like. Static electricity appears to do good when employed for a long time, especially when combined with hydrotherapeutic measures and isolation.

If the patient be so tormented by fixed ideas as to be practically mad, the strictest guard should be kept over him.

CO-ORDINATED TICS.—As was said in an early part of this article, this variety of tic differs from the simple tics in the more complex character of the movement. Many of the cases properly included in this group are instances of habit-movements which cannot be regarded as disease. The tic may be no more than some act of every-day life, repeated time after time without obvious cause.

The case mentioned by Sir William Gowers, of a boy who was in the habit of bringing both his arms forward and then stooping, belongs to this group; as does another, in which a girl, when walking, would make a half-turn, as if looking for something that she had dropped.

Osler refers to a child who was recovering from what was supposed to be chorea minor, and who first smelt and then blew on anything she took into her hands. The same observer described a boy, the subject of facial tic, who used to bite his middle finger hard, and at the same time press his index finger against the point of his nose.

Sinkler has also recorded some cases of the same kind—such as that of a girl who, when walking, would stop, rub the toe of her shoe against the calf of her other leg, and then go on as if nothing had happened. And he refers to a boy who, as the result of imitating the motor man of a trolley car, acquired the habit of standing with his back to a door and swinging round first one and then the other arm, and giving vent to an explosive utterance.

It happens not uncommonly that such a habit-movement or trick may be controlled more or less by an effort of the will; so that as long as the patient keeps the matter in mind he may be able to prevent its recurrence, while as soon as he forgets about it the movement occurs automatically, perhaps without his perception of it. In such cases continual efforts to control the movements may result in a permanent cure. Thus a special feature described in connection with the last variety of tic may be wanting, that is, the impulsive tendency to execute some movement which the patient is quite conscious of, yet which he is unable to prevent. The more usual movements which characterise simple tic may coexist in the same individual with those of a more complicated character.

Noir, in Bourneville's clinic at the Bicêtre, has admirably described the co-ordinated tics which are met with in feeble-minded children. Among the more common movements in such cases are balancing, jumping, rolling the head from side to side, striking the chest with the chin, and hitting the head or chest with the hand. The latter habit is what Rubinowitch has called "Krouomania," and, like the other movements, these are sometimes repeated in a rhythmical manner; the balancing, nodding, and rotation movements are more especially likely to preserve a definite rhythm. Or the subject may stoop from his chair, stretch himself out prone on the floor, raise his arms above his head, and so forth, all of which actions are performed from time to time in orderly sequence.

Osler refers to a case at the Institution for Feeble-minded Children at Elwyn, in which an idiot (known as the "Dervish"), after rising from the floor, balancing, and making a few sweeping rotations, would execute a series of revolutions in which with his arms spread out, and moving but little from one spot, he increased the speed of the rotations until he spun round like a top, and finally fell exhausted.

Some of the cases included by Noir in his account of tic occurred in blind subjects, in whom quick movements of a finger in front of the eyes were especially common. Noir considers that there is probably some perception of light in such cases, and that the tic is the outcome of the habit of producing shadows by movements of the finger in front of the eyes.

Prof. Osler is disposed to regard the affection known as "head-nodding" (see p. 903), which occurs in infants, and has been fully described by the late Dr. Hadden, as belonging to the group of co-ordinated tics; and he considers that the disorder described by Dr. Gee as "Head-banging" comes also within the same category. The latter condition was met with in three children, two of whom were two and a half years old; in one of these the head-banging had occurred for two or three months, in the other for six months; the third child was five years old, and had been affected for two and a half years. The younger brother of one of these patients acquired the trick when put to sleep with the one affected; but, when separated from him, he lost it. These children were in the habit of turning on their faces in bed at night, either when awake, or possibly half awake, or when sound asleep, and would bang their heads into the pillow several times in succession. In one of them the bangings occurred about six times only, and was seldom repeated the same night, or more often than one night in four; but in one of the others the banging went on sometimes nearly all night, and the child awoke very weary in the morning. The condition was not accompanied by any other symptoms, nor was there any evidence of its kinship to epilepsy or any other such disorder.

Similarly, Osler regards as an exaggerated example of co-ordinated tic Weir Mitchell's case of the man who, unless completely at rest in the recumbent posture, would strike his side by a pendulum action of his left arm; this movement he repeated about 150 or 160 times a minute, in regular order.

PSYCHICAL TICS.—There is but little to add to what has been said already on this subject under the head of convulsive tic. A psychical tic has a like origin, and is equivalent to the motor variety. In the latter a spasmodic movement occurs which the subject is unable to resist; in the former an irresistible impulse compels him to emit a certain word, perform a certain act, count a certain number before doing something, and so on.

In some instances the imperative conception is generated by some external stimulus or antecedent thought, while in other cases it seems quite spontaneous, and cannot be traced to any such cause. At times after the discharge there is temporary relief, but in other instances the victim, after a long struggle between volition and the irresistible impulse, ending in the victory of the latter, is left much exhausted.

I have already indicated that the motor and psychical tics may concur in the same subject, and that the former may occur without the latter; it now remains to be said that psychical tics may occur without motor equivalents. Moreover, as a motor tic may be of no serious moment, so a psychical tic may be equally insignificant; imperative ideas may persist for years, and, although often a source of annoyance to the victims, no more serious mental disturbance may result: as has been more especially emphasised in this country by the late Dr. Hack Tuke.

The thoughts which incessantly recur are usually both foreign and unwelcome, and words which the victim feels compelled to utter are neither in conformity with his custom nor what he wishes to pass his lips; so that if they escape it is in spite of his efforts to keep them back. Those who always touch certain objects which they habitually pass may do so automatically, as a sort of habit; or this proclivity may be related to the mania for counting (arithmomania); or the patients may be impelled to the act by a dread that something undesirable will happen if they omit their observance. This is a common enough trick of children, and one usually of no consequence, even if it persist, as is sometimes the case, until adult life. No better example of this condition is to be found than the well-known one of Dr. Johnson, who, as he walked along the street, used to place his hand on certain posts; if he missed one, he would turn back and perform the accustomed ceremony before proceeding on his way. The antithesis of this *délire du toucher* is the dread of touching certain objects, a condition spoken of as mysophobia. In the case of a doctor, referred to by Hack Tuke, the imperative idea was that something serious would happen if he trod on the divisions between the flags of the pavement, so that in spite of being fully alive to the absurdity of the notion, he avoided them.

Arithmomania, or the irresistible impulse to count a certain number before doing a thing, is well illustrated in the case of a girl, recorded by Osler, who had to tap on the edge of the bed nine times before getting into it; to count a hundred after brushing her teeth; to knock three times on a window by the side of the door, and a similar number of times on the door itself, before she would unlock it. The tendency in these cases of arithmomania may be to make endless calculations, to count certain letters in words, the number of books on a table, and so on.

Then there is the state in which a certain name or word is sought for with an intensity that becomes painful, and in which, when found at last, the person is impelled to repeat time after time (onomatomania). It may also manifest itself as an attribution of a disastrous significance to certain words.

What has been called the "*insanity of doubt*," or "*maladie du doute*," forms an important class of imperative ideas, a good example of which is Dr. de Watteville's case, related by Hack Tuke. The patient always had a feeling that he had done a thing wrongly; for instance, after posting a letter or putting something into a drawer, he had grave misgivings as to the accuracy of what he had done. So, too, on alighting from an omnibus he would begin to think that he had left something behind him. Although fully aware that there were no grounds for his misgivings, yet the ideas tormented him.

Both agoraphobia and acrophobia are common forms of imperative ideas. The former condition makes it well-nigh impossible for the affected person to venture across any large open space owing to the fear that something untoward will befall him; in the latter state the subject finds it difficult to resist a strong impulse to throw himself from any height, in spite of his being fully aware of the unreasonableness of this impulse. Neither giddiness nor suicidal intent plays any part in such cases. The condition of mysophobia, already referred to, belongs to the same class of fixed ideas, many other varieties of which might be mentioned, though to do so would not serve any very useful purpose. The fundamental characters of imperative ideas have been sufficiently set forth to allow the reader to multiply examples for himself at will.

As was said when convulsive tic was under consideration, no hard and fast line separates the mental state characterised by fixed ideas from that of true insanity, so that at any time the border may be crossed, and what was formerly recognised by the patient as absurd now becomes a delusion in which he firmly believes.

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PARAMYOCLONUS MULTIPLEX

SYN.—*Myoclonus; Multiple myoclonus; Myoclonus epilepticus; Myokimie; Myospasia Simplex; Spinal epilepsy; Friedreich's disease; Convulsive tremor.*

THIS affection is a motor neurosis characterised by sudden shock-like clonic contractions, usually of corresponding muscles on the two sides of the body; the spasms may not only be symmetrical, but also isochronous.

The affection was isolated from the chaos of motor neuroses by Friedreich in 1881, the condition is therefore sometimes designated by his name; but this observer's name is more familiarly associated with "Hereditary ataxy" (p. 152).

As Friedreich's description was taken from a single case, it was quite impossible for him to draw a clinical picture applicable to all such cases; for a neurosis of the kind must vary in intensity, and in the extent to which different parts of the body are involved. Many indubitable instances of this condition have not conformed in all respects to the clinical picture presented by Friedreich. On the other hand, from 1883 to 1890, a number of cases were published as examples of this affection, many of which at any rate are to be referred to other related motor neuroses, or to hysteria.

In 1891, however, Unverricht was able to give a much wider conception of the affection in his monograph entitled "Ueber Myoclonie"; his description was based on five cases personally observed; and further he criticised the records on the subject collected between the time of Friedreich's publication and his own. I see no reason to distinguish the affection described by Friedreich as paramyoclonus from that described by Unverricht as myoclonus; and I preserve the name applied to it by the former observer as more comprehensive.

In searching through the records of motor neuroses prior to Friedreich's publication, it is impossible to avoid being impressed by the similarity between the condition named by him and one to which attention was first called by Sir Russell Reynolds; this latter case was one of epilepsy in which, in the intervals between the attacks, there occurred sudden shock-like contractions of various muscles. This resemblance has been emphasised of late, since it has been shown how frequently myoclonus and epilepsy are associated in the same individual. In the case described by Sir Russell Reynolds the clonic muscular contractions were unattended by loss of consciousness, although sometimes they were violent enough to throw the patient to the ground.

This condition has been described by some writers as "spinal epilepsy," a designation to be avoided, for this appellation has long been identified with the clonic spasms which occur in the parts below a destructive lesion of the spinal cord.

It is questionable whether the condition described by Hammond as "Convulsive tremor," and regarded by himself and other American observers as identical with Friedreich's paramyoclonus, has any real claim to be so regarded.

Causation.—Little definite is known of the etiology of the affection. Direct heredity does not appear to have been traced, except in rare instances; and in many cases there is no indirect heredity in the shape of other neurotic affections in the parents or grandparents of the patients, though in some of them an indirect heredity appears. In not a few there has been a history of alcoholism in one of the parents.

That the affection may be congenital in the sense that the tissues become susceptible to it during uterine life, although the disease does not manifest itself for a certain interval after birth, is suggested by cases such as those recorded by Unverricht, in which five children of the same family became affected between the ages of six and thirteen years. Whether such cases prove a congenital taint or not, they prove that the affection may run in a family. The possibility of simulation is made improbable by the fact that in one child, which was brought up away from the family, the disease appeared, nevertheless, at the time of life when it had broken out in the other members.

Moreover, the association with epilepsy in Unverricht's case lends support to the view that the condition was genuine.

The tendency for the affection to manifest itself where there is a degenerative condition of the nervous system, whether congenital or acquired, is shown by its frequent association with epilepsy—a disease in which certain tissues are prone to degeneration, or are actually undergoing this change.

That shock, mental or physical, may set up this spasmodic disorder, as in Friedreich's patient, there can be no question; but it is equally certain that no such exciting cause may be needed to evoke it; many cases have been recorded in which this cause was absent.

Again, the myoclonic spasm may manifest itself in the weakness of overwork or acute illness, as in several instances after severe and repeated attacks of malaria. It has been looked on, by Lemoine and Lemaire, as related to neurasthenia.

Symptoms.—According to Friedreich's original description, the following are the characteristics of this motor neurosis; namely, clonic spasms in a number of symmetrical muscles of the upper and lower limbs, interfering in no way with motor power and co-ordination of movement, ceasing during sleep and during voluntary movements. The locomotor effects of the spasms is either nil or but very slight, and the muscles of the face and trunk escape. The nutrition of the affected muscles is unaffected, and their mechanical and electrical irritability is unaltered. In his case there was no affection of sensation, the superficial reflexes were increased, and there was also marked increase of the knee-jerks.

The case which enabled Friedreich to give this description of the affection was the result of a severe shock, and the spasms, after lasting several years, rapidly ceased.

Excellent as is his description, and applicable as it is to some cases of the affection, it requires considerable modification when we attempt to regard it as a type of the class of cases that ought in my opinion to be included in the same category as that of Friedreich, for they differ from it in several important particulars. In the first place, the intensity of the spasms differs considerably in different cases, so that in some the locomotor effect may be great, in others little or nothing. So, too, the extent of the invasion of different parts of the body varies; and both the face and trunk muscles, which escaped in Friedreich's case, may be involved. Moreover, voluntary movement has by no means invariably an inhibiting effect on the spasms; on the contrary, in some cases such movements evoke the spasms, or intensify them. Then again the knee-jerks, which in Friedreich's patient were markedly increased and are often in excess, may in many cases be normal or even diminished. Lastly, the favourable termination of Friedreich's case is by no means usual; and indeed in this very case the recovery was only of short duration, for, according to Schultze, the patient quickly relapsed, and the spasms never ceased again till his death.

These preliminary remarks will clear our view of the class of cases to be included in this affection, whether we hold to the name originally given to it by Friedreich, or whether we follow Unverricht, whose appellation, though shorter, is less comprehensive. With the broader conception of the affection which I have indicated above, we are now in a position to consider the various symptoms of the malady more in detail.

The spasmodic movements are lightning-like in rapidity, each contraction being a shock, and so sudden as to be exactly like the shock which is obtained from a muscle by a single faradic excitation. Usually the sudden contraction in one muscle is followed by an equally sudden contraction in another; in other instances the spasm is repeated in the same muscle several times in succession. In the latter case the clonic spasm may sometimes seem to pass into slight tonus of very short duration; but even so slight a tendency to tonic contraction is rare, the muscular spasms being conspicuously clonic. There is no rhythm in the succession of the shocks; they may occur singly or in batches separated by quiet intervals; as many as forty or fifty clonic contractions may occur in the course of a minute.

The spasms may seize muscles singly, parts of muscles, or whole groups of them; but the great characteristic of the distribution of the spasm is that it attacks muscles which are not supplied exclusively by a single nerve, or which cannot possibly be thrown singly into contraction by volition; even when a group of muscles is involved simultaneously in spasm the combination is without any co-ordinated function, and is such as no effort of the will can reproduce.

Bastianelli considers that there is a special group of cases of myoclonus characterised by fibrillary contractions of the muscles, in which those of the face may participate. He states that in these cases the electrical excitability of the muscles is sometimes increased; and there is always an unduly persistent contraction after the excitation has been discontinued. Paræsthesiæ and paralgesiæ form part of the clinical picture of this group, to which Giovanni Biancone has given the name "Myokimie." According to this observer only five such cases are on record: his own, Bastianelli's, and others by King and by Schultze.

Another characteristic feature of the distribution of the spasms is that they may be strictly symmetrical on the two sides of the body. Not only may the muscular spasms be thus strictly symmetrical, but they may also occur simultaneously in the same muscles on the two sides. The latter is by no means an invariable rule, however; for instead of being isochronous, the spasm on one side may be followed by contraction of the same muscle on the opposite side after an interval which indeed is usually of very short duration. Symmetry is not invariably observed, however, for, in exceptional cases, one side of the body has been affected alone, as in a case described by Chauffard.

Certain muscles are much more commonly concerned than others, although in any given case nearly the whole of the voluntary muscles of the body may be affected, with the exception of the ocular muscles, which are always spared. The muscles of the limbs are affected more frequently than those of the trunk, and the latter more commonly than those of the face; and, as we have already seen, the muscles both of the trunk and face escaped in Friedreich's case, those of the limbs being affected alone. The facial muscles were free from spasm in about half the recorded cases. All the muscles of the limbs are not equally prone to the affection; the upper are more commonly affected than the lower limbs, and the spasms usually seize the muscles about the shoulder girdle and upper arm, and those of the thigh, while the muscles of the forearms, hands, legs, and feet more commonly escape. Thus it is the proximal segments of the limbs that are most affected; the distal segments are always much less so, while the most peripheral parts remain quite free from spasm.[1] The pectorals and deltoids are especially liable to be affected; in the legs the quadriceps extensor group, and, in the trunk, the abdominal recti, obliques, and the erector spinæ. But even the diaphragm and cremasters may share in the spasms.

The extent to which any locomotor effect is produced by the spasms is a very variable quantity. In Friedreich's case, the absence of any locomotor effect of the muscular contractions was regarded as one of the characteristic features of this motor neurosis, a feature which is shared by many cases of the affection. Indeed the muscular spasms may not be observed until the patient's clothes are removed, so as to reveal the muscular contractions. On the other hand, the sudden and severe shock-like contractions may throw the patient to the ground, and that, be it observed, without the slightest affection of consciousness. Between these two extremes we meet with all degrees of locomotion. Thus there may be a sudden shrug of the shoulders with an equally sudden drop; or they may be drawn forwards or backwards; or the forearm may be slightly flexed on the upper arm and then extended; or the movement may be one of pronation or of supination; more rarely of flexion or extension of the hand at the wrist, or of the fingers; or the latter may be separated or adducted. The spasms in the muscles of the lower limbs may result in rotation, flexion, or extension; in stamping of the feet; in swaying of the trunk from one side to the other; or in its being jerked forward or backward, displacements which may also be brought about by contraction of the trunk muscles themselves.

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[1] In a young man, a well-built but rather delicate-looking undergraduate, who consulted me for myoclonus about 20 years ago, the spasm was confined to the sartorius. When I saw him this muscle in the left leg was affected alone, and such was, I believe, the usual state of things. But, if I remember aright, this muscle of the other leg was convulsed at times. The limb was quite stationary during the spasms. During my observation the patient was standing. My impression is that no other muscles were concerned, but unfortunately my notes of the case are destroyed. There was a terrible history of alcoholism in the family, and to this craving, I understand, my patient in his turn fell a victim at a later date. Otherwise the members of the family were big, vigorous persons with no morbid proclivity. However, I am under the impression that my patient had had rheumatic fever not long before the spasms appeared. I was unable at that date to find any record of the spasmodic affection.—ED.

Attitude appears to have a distinct influence on the spasms; for, as a rule, they are less pronounced when the patient is standing than when sitting, and when sitting than when lying recumbent; a state of things no doubt closely associated with the next problem, namely, that of the influence of voluntary movements on the spasms. This we may consider from two standpoints—the influence of the former in inhibiting, accentuating, or evoking the latter; and the degree in which the latter interferes with the former.

It will be remembered that one of the characteristic features in Friedreich's first case was that voluntary movements inhibited the myoclonic spasms, so that all such spasms ceased during the performance of a voluntary act. This has been the case in other recorded cases of myoclonus; but it is as common to meet with cases in which, instead of diminishing the spasm, voluntary movements actually augment it, or even evoke the involuntary spasms in parts quiescent up to the moment of the volition. Similarly, any form of mental excitement or physical fatigue will usually augment the spasms. In some cases all spasms are in abeyance so long as the patient remains quiescent; while in others the spasms go on quite independently of repose, and may indeed, as we have already seen, be more marked in the recumbent than in any other posture. In nearly all cases the spasms cease entirely during sleep; though in one, recorded by Schütte, although the more pronounced clonic shocks ceased, fibrillary contractions persisted.

Voluntary movements are not generally interfered with by the myoclonic spasms, even when the former evoke or intensify the latter; but in the more severe cases, with more pronounced locomotor effects, ordinary voluntary acts are of course a good deal interfered with. Apart from such interference, however, diminution of motor power is no part of the type; nor is there any affection of co-ordination of movement, other than interference with a voluntary act by the unexpected intrusion of some unbidden muscular contraction.

Mechanical excitability of the muscles is usually increased; and percussion of a muscle, or irritation of the skin, may suffice to evoke myoclonic spasms.

The electrical excitability of the affected muscles is unaltered, but the application usually intensifies the spasm; in some instances an undue persistence of the contraction of the muscle after shutting off the current has been noted.

Myoclonus and epilepsy.—This association was present in Unverricht's cases; and the number of recorded cases in which the two spasmodic affections have occurred in the same individual is rapidly on the increase. Two instances of the kind were shown at a meeting of the Neurological Society of London by Dr. Ferrier in 1897; the patients were brothers, and the proposal was made to designate such cases "myoclonus epilepticus,"—to distinguish them from cases of myoclonus unassociated with epilepsy. The epileptic attacks commonly occur at night, and it is not unusual to be able to foretell a fit, as the myoclonic contractions become much worse during the period immediately preceding it. So, too, the spasms may be more intense for a time after an epileptic fit: in other instances, however, they are considerably less marked after the epileptic attack.

Speech may be interfered with by the spasms in different ways: thus a sentence may be interrupted, and then after the pause be continued normally; or the patient may pause to try to stop the spasm; or spasm of the tongue or muscles of the throat may cause the interruption. On the other hand, a word or syllable may be cut off by an unexpected spasm; or, as muscles of the trunk suddenly contract, or those concerned with expiration, a word may be blurted out.

Sensory phenomena.—If we except the class of cases which Biancone proposes to call "Myokimie," in which subjective sensory disturbances constitute an important part of the morbid group, there is, as a rule, a total absence of sensory phenomena in myoclonus. There are no paræsthesiæ, as a rule; and the muscular contractions, though in their violence and persistency often fatiguing, do not give rise to any pain, and there is no blunting of sensation. There may, however, be a stiff feeling in the muscles, and pain in the back; and wandering pains in the limbs, have been noted. Romberg's sign has been present in some cases.

Reflexes.—The superficial reflexes are commonly increased, as in Friedreich's case, though not necessarily so; the tendon jerks may be either unaltered, increased, or diminished. The sphincters are unaffected.

Psychical disturbances.—Mental disturbances are exceptional, although Raymond regards the intellectual faculties of the subjects of this disease as rarely quite normal. In a case recorded by Stembo there was some psychical disturbance, which manifested itself by verbal explosions which the patient was unable to prevent, and which resembled the condition

met with in convulsive tic (p. 877). In two cases observed by Bresl
there was progressive enfeeblement of intelligence, and a fatal issue.

Trophic disturbances.—As a rule there are no trophic disturbances; bu
cyanosis of the extremities, profuse sweating, and similar vaso-moto
troubles have been met with exceptionally.

Thoracic and Abdominal organs.—An excess of indican has been presen
in the urine in some cases, and in greatest amount on the patient's ba
days; but many cases of paramyoclonus, otherwise typical, show no suc
increase of indican.

Diagnosis.—The affections with which paramyoclonus is likely to b
confused are chorea, electric chorea, convulsive tic, and hysteria.

Chorea.—As regards the diagnosis between paramyoclonus and ordinar
chorea there ought, as a rule, to be no real difficulty. The spasmodi
movements in chorea resemble voluntary movements, and are the resul
of contractions of groups of muscles in synergic combination. How fa
this is from being the case in paramyoclonus I have already shown. Then
again, voluntary movements usually evoke the movements of chorea, wherea
in paramyoclonus it commonly happens that the involuntary movement
are inhibited by volition. Furthermore, in chorea the movements ar
commonly unilateral; in paramyoclonus symmetrical affection of simila
muscles on the two sides is the rule, unilateral affection being a
extremely rare event. We have already seen that the face is affected
only in about one-half the cases of paramyoclonus, whereas facial grimaces
are among the most common manifestations of chorea.

Electric chorea.—If by electric chorea we mean the affection described by Dubini (*vide* p. 864), there should be little difficulty in diagnosis, in spite of the occurrence of epileptic convulsions; Dubini's disease is attended with high temperature, progressive paralysis with muscular atrophy, and loss of faradic excitability of the muscles; it runs an unfavourable course and terminates in death. There is much more likelihood of mistake in the cases of ordinary chorea in which the spasmodic movements are so sudden and rapid as to have suggested the name of electric chorea; but, in spite of this resemblance in the rapidity of the contractions, attention to the points indicated in the diagnosis of ordinary chorea should serve to distinguish this variety also. In the hysterical affection which has been called electric chorea the presence of the usual stigmata of hysteria are present, as a rule, and serve to distinguish it; moreover, as in chorea, the contractions are of synergic groups of muscles, such as can be brought into action by an effort of the will, not of isolated muscles or of parts of these. The condition described by Henoch as electric chorea appears to be none other than a form of tic or paramyoclonus; and in one of his cases the condition was associated with epilepsy.

Convulsive tic.—If by this is meant facial spasm there is no possibility of confusing the two conditions; for in paramyoclonus the face is commonly spared, and when affected it is only as a part of a more general exhibition of spasm. Moreover, facial spasm is commonly unilateral, and even when it becomes bilateral one side of the face is usually more markedly affected than is the other. The clonic spasms become more and more rapid during the attack until, when at its height, they may so run into each other as to result in tonus; then, as the attack is passing off, the clonic spasms become slower and slower until they ultimately cease. The spasms are restricted, as a rule, to the distribution of the facial nerve, a restriction foreign to paramyoclonus as regards any nerve-supply; they are evoked by voluntary movement or emotion, and are never inhibited by the former as is so often the case in paramyoclonus.

If, on the other hand, by convulsive tic is meant the generalised affection —the "maladie des tics convulsifs" of French writers (p. 873)—then there is more likelihood of confusion. In this condition, however, there is a peculiar mental state, and the face participates in the spasms which take the form of gestures; there is an impulsive tendency which the patient cannot resist, and which may be exhibited by the imitation of some gesture (echokinesis), the repetition of words (echolalia), or a tendency to blurt out obscene words (coprolalia). From this it will be obvious that there is little real similarity between the two neuroses; but examples of connecting links, as it were, between the two affections may cause a little difficulty at times, as in the case of a patient, like that observed by Stembo, in whom the myoclonic spasms were associated with an irresistible tendency to ejaculate certain words and sentences.

Hysteria.—Probably the most difficult problem in the diagnosis of paramyoclonus is its distinction from hysteria. Indeed, in some cases, if we are to admit that cases like that recorded by Hoffmann are in reality cases of hysteria in the guise of paramyoclonus, the distinction becomes well-nigh impossible. For without the presence of some of the usual stigmata of hysteria to assist us such a case presents no differences from the group which has been separated from the other motor neurotic groups and designated paramyoclonus. When epileptic attacks occur the diagnosis is made certain. But, apart from this, if we attribute to hysteria only such spasmodic movements as can be reproduced by an effort of the will (the proper position to take in the matter, it seems to me), then the distinction between the spasms of hysteria and those of paramyoclonus at once becomes evident. Moreover, in hysteria it would be unusual not to find some of the stigmata of this affection in addition to the spasmodic disorder.

Prognosis.—The affection is usually slowly progressive up to a certain stage; then it may remain stationary for many years, and the patient may ultimately die without any period of freedom from the spasms. In exceptional cases it has been said to run a rapid course, and to end fatally within a few months of the time of onset. Spontaneous recoveries, or recoveries under treatment, have occurred; but, on the other hand, relapses are common, so that when the affection has once existed it is apt to recur; more especially if the patient be again exposed to the influence of what seemed to be the immediate cause of the first attack.

Pathology.—*No changes have been found in the neuro-muscular system to account for the condition.* Certain authors, notably Möbius, Strümpell, Hirt, and Pitres, attribute myoclonus to hysteria; others, while admitting that there is a motor neurosis distinct from hysteria to which the appellation myoclonus or paramyoclonus is applicable, nevertheless consider that hysteria may present itself under the guise of myoclonus. Thus Hoffmann has recorded a case, which he regards as of this nature, occurring as the result of a shock in a girl, sixteen years of age, who had had a similar attack four years previously. Bötticher, who recognises two principal types of the affection, attributes the paramyoclonus of Friedreich to hysteria, while regarding the myoclonus of Unverricht as identical with Huntington's chorea (p. 859).

That hysteria should manifest itself in a manner more or less resembling the clinical picture of a myoclonus is scarcely surprising, when we remember how closely hysteria may simulate the manifestations of other affections of the nervous system; and especially when we remember further that paramyoclonus itself is a functional motor neurosis. But the following considerations, among others, indicate that all cases of paramyoclonus cannot be hysterical.

Perhaps nothing is more opposed to the hysterical hypothesis than the association of myoclonus with epilepsy. Again, in the present malady there is little evidence of the concomitance of any of the usual stigmata, one or more of which we expect to find in a subject of hysteria. Moreover, were the condition hysterical, it would be not a little strange that men should be more commonly affected with myoclonus than women. While admitting, then, that hysteria, in the character of its manifestations, may resemble paramyoclonus, there appear to be good grounds for discarding the hysterical hypotheses, as inadequate to account for this kind of motor neurosis.

Another conjecture is that myoclonus belongs to the group of chronic choreas. It is not improbable that a good many cases recorded as "electric chorea" belong to the group now under discussion. I have already pointed out that some confusion has arisen in the use of the name "electric chorea" for some cases of ordinary chorea in which the muscular contractions have been unusually sudden; whereas others reserve the name for the so-called Dubini's disease. The chances of confusing Dubini's disease and myoclonus is the greater as both may be associated with epileptic attacks; but the loss of motor power attended with atrophy of the muscles and loss of faradic excitability which form part of the clinical picture of the former affection serve to distinguish it from myoclonus.

I have already compared paramyoclonus with "electric chorea," with the "maladie des tics," and with Huntington's chorea.

A third view finds favour with Lugaro and others. According to them the muscular spasms in myoclonus are the "exteriorisations," so to speak, of a general myoclonic state of the neurons of the central nervous system; a state that may find expression in the fibrillary trembling of neurasthenics, fibrillary chorea, or paramyoclonus of Friedreich, when the neurons of the anterior horns of the spinal cord are the seat of the morbid change; in electric chorea and common tic, when the motor neurons of the second order are involved; and "maladie des tics," when the psychomotor neurons are implicated.

And, lastly, must be mentioned Wagner's suggestion that we should perhaps look to the thyroid for an explanation of the condition, as the muscular spasms of animals after extirpation of the thyroid resemble those of paramyoclonus; and, indeed, that in a case of his such spasms were cured by thyreoidin.

There is an equal want of unanimity of opinion as to the part of the neuro-muscular system that is at fault. Of the different views advanced, that of Popow, which supposes the defect to be in the muscles themselves, may be dismissed at once. The points on which Popow bases his opinion are some of them erroneous, while others are too speculative to call for serious consideration.

Friedreich's view, subsequently supported by Unverricht, that the seat of disturbance lies in the anterior horn cells of the spinal cord, has many adherents, including Lowenfeld, Marie, Ventick, and others; and one of the strongest arguments in its support is the symmetrical distribution, and in some cases the isochronicity, of the myoclonic spasms. Against this view Minkowski and others have urged that the muscular spasms are essentially clonic, whereas spasms of spinal cord origin are tonic in character; but Unverricht points out that the neurons of the brain and spinal cord may both be concerned in tonic or clonic spasms, and that clonic spasms may derive from the spinal cord even after it has been separated from the brain. Turtschaninow has succeeded in evoking clonic muscular contractions experimentally in dogs, the spasms resembling the myoclonic contractions observed in Unverricht's cases; and he found that the contractions continued even after ablation of the cerebrum, section of the cerebral peduncles, or transverse section of the spinal cord high up.

Most of those authors who have opposed the supposition that the spinal cord is the seat of the disturbance, have advanced, as an alternative hypothesis, that derangement of the motor neurons of the cerebral cortex is concerned in the spasms; among them may be mentioned Rubino, Seppilli, Minkowski, Grawitz, and Raymond.

It has been urged that the frequent association of epilepsy with myoclonus is a strong argument in favour of the cerebral origin, as the former disease is essentially an affection of the cortex cerebri. So, too, the fact that the myoclonic spasms are increased before and after the epileptic attacks has been regarded as additional evidence pointing to the probable dependence of the two conditions on some defect in the same part of the central nervous system. As I have said, the clonic character of the spasms has been regarded as another point in favour of the cortical basis of the affection; and it has been stated further by some writers, such as Raymond, that, besides the influence of moral causes in evoking or exaggerating the spasms, the intellectual faculties of the subjects of myoclonus are rarely quite normal. The presence of athetoid movements and hemianæsthesia, in a case observed by Minkowski, is also regarded as supporting the cortical origin. The chief points urged by Unverricht and others against the attribution to the cerebral cortex are that the spasms are symmetrical and often isochronous, that they involve single muscles or parts of muscles, and that even when groups of muscles are simultaneously involved they are not such as act together in synergic combination; whereas in all spasms known to be of cerebral origin the muscles involved are those which are brought into action synergically, and which result in co-ordinated movements like those of volition; so that flexion of the elbow, for instance, may be a consequence of the contraction of the group of muscles concerned in bringing about this movement, but that isolated contraction of a muscle, as for instance of the supinator longus, is never met with. It has further been pointed out that the most delicate electrical stimulus applied to the cerebral cortex can only evoke contractions of a group of muscles concerned in some particular movement, and never of an isolated muscle.

The last suggestion that we have to consider is that, according to the phenomena, the motor neurons both of the brain and spinal cord may be affected; and that the neuroclonic state existing in the neurons of the anterior horns of the spinal cord or their homologues in the medulla oblongata and pons, may give rise to myoclonic spasms; whereas an altered state in the neurons of the cerebral cortex may be subsequently and secondarily induced, giving rise to epileptic attacks. Or again the altered condition of the cerebral cortex, and the consequent epilepsy, may be primary, and the neuroclonic state of the neurons of the spinal cord may appear subsequently.

In the present state of our knowledge of this subject all such arguments are of necessity largely conjectural; nevertheless, there seems much to recommend the last hypothesis. Indeed, it seems to me that, in a manifestation of a degenerative condition of the central nervous system, the degenerative changes in the neurons of the cortex, depriving them of sufficient control of the spinal neurons, may be, in part at least, responsible for the neuroclonic state in those of the spinal cord; whether they themselves are so altered as to give rise to epileptic fits or not.

Treatment.—It is naturally of primary importance that measures should be adopted to improve the general condition, and to bring about a more stable condition of the motor neurons of the central nervous system by improving their nutrition. Although the affection manifests itself so often without any evidence of an exciting cause, such should always be sought for, and if still found in action should be promptly dealt with; for it must be remembered that the longer some perverted action of the motor neurons continues the more difficult it becomes to break them of a habit established by frequent repetition.

Cures have been credited to the application of the constant current along the spinal column; indeed it has been asserted that this mode of treatment rapidly brings about recovery. Three points must be borne in mind, however: some patients recover spontaneously, some are possibly instances of hysteria in the guise of paramyoclonus, and, lastly, supposed cures have proved to be but temporary cessation of spasms which have returned after a short interval, and have persisted up to the time of the patient's death: this was the case with Friedreich's patient.

Hydropathic methods have also found favour in the treatment of myoclonus, either alone or variously combined with electrical treatment.

Of drugs, arsenic, given in large doses, has the reputation of procuring recovery in some cases. Chloral controls the spasms temporarily, but when its use has to be long continued it loses its effect. The bromides have much less influence over this condition than over epilepsy.

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SALTATORY SPASM

SYN.—*Saltatoric spasm ; Static reflex spasm.*

THIS is a rare affection in which clonic contractions of the muscles of the legs are evoked directly the subjects place their feet on the ground, with the result that the patient is raised a variable distance in the air and then let down again; the same thing being repeated time after time as long as the erect posture is maintained.

Bamberger described the first two cases of this variety of spasm in 1859; and similar cases were published subsequently by Beigel, Guttmann, Frey, Gowers, and others; but the total number of recorded cases is small.

Both sexes are prone to the affection, though males appear to be attacked a little more frequently than females. Cases of the kind have been met with at different ages between ten and seventy years. Prominent among the etiological factors, in the majority of cases, is debility induced by disease, old age, or certain excesses; in all of which states depression of the nervous system is apt to occur. In a minority of cases epilepsy, hysteria, and other functional disturbances of the nervous system have preceded the appearance of these spasms, while in some cases the spasms seem to have been a manifestation of hysteria. In one case a convulsive seizure was the immediate precursor of the saltatoric spasms. Apart from the condition following immediately on some debilitating illness, as in one or two cases, no immediate cause can, as a rule, be traced. In Frey's case the saltatoric spasm was really a symptom of chronic myelitis; paresis, muscular atrophy, and contracture formed part of the clinical picture.

The onset is usually abrupt, though in exceptional instances premonitory symptoms have been met with, such as stiffness in the legs or a subjective sensation of it, a sense of weight, or tearing pains; in other cases slight tremor has been observed.

The essential feature of the affection consists in alternate contractions of the flexor and extensor muscles of the legs, which may be sufficiently violent to throw the patient repeatedly into the air,—even to the ground unless he happen to be supported. In these or various jumping or hopping movements the feet leave the ground; but when the spasms are less severe the heels are drawn up, while the toes remain on the floor. I have said that the spasms are induced directly the sole of the foot touches the ground, and are repeated in rapid succession as long as the erect posture is maintained. In the sitting or recumbent postures there is usually a complete cessation of all spasm, though slight rigidity or clonic spasm may persist for a short time; yet even in these positions the spasms can, as a rule, be evoked in slight degree by pressure on the soles of the feet. Muscles of the lower limbs other than those which act on the ankle joint may be involved, for in one of Sir William Gowers' cases those acting on the knee and hip were thus affected. The distribution of the spasm in different cases varies a good deal; so that, although the spasms described in the legs are the chief character of the affection, muscles of the back, face, neck, and even of the pupils have been concerned in some cases. Thus in Bamberger's second case opisthotonos was produced by the contraction of the trunk muscles; in his first there was spasm of one side of the face, and the pupils were alternately dilated and contracted. The spasms induced by standing do not spread to the arms, but slight spasms have been observed in the muscles of the arms when certain voluntary movements were performed by the patient in the recumbent posture. Mental influences cause an exacerbation of the spasms in some patients, in others arrest of the spasms is thus brought about.

The spasms usually continue for several months; exceptionally the duration has been less than a month; in one patient the duration was only a few days, in another they continued up to the patient's death, six years after their first appearance. Recovery is usually gradual, and the spasms may recur after an interval of perfect freedom.

Motor power is unaffected, as a rule, but a very slight degree of enfeeblement has been noted in some cases after the spasms had been in existence for some time. Tenderness of the spine has been found in some cases; and subjective feelings of dull pain in the legs, without cutaneous hyperæsthesia or anæsthesia, has been noted. In addition to the peculiarities already described in one of Bamberger's patients, dyspnœa and palpitation also were present.

In the case in which the spasms continued for so many years death was the result of enteric fever; in a case recorded by Kollmann death resulted from other severe disturbances of the nervous system.

All the evidence at our disposal points to the spinal cord as the seat of the morbid changes. Moreover, it would seem that the abnormal irritability concerns especially the centres of the co-ordinated movements of jumping. It is interesting to note here that Freusberg determined that in animals alternating flexion and extension of the legs can be produced in the lumbar enlargement of the spinal cord. Woroschiloff, on the other hand, found that, in the rabbit, a centre concerned with the co-ordination of springing movements of the legs exists in the upper part of the spinal cord.

Freusberg's results obtain support from an observation by Sir William Gowers in man, in a case of paraplegia with evidence of a transverse lesion in the thoracic region of the cord with complete motor paralysis; in this patient pressure on the soles of the feet evoked alternate flexion and extension at the hip, knee, and ankle joints, so that the feet were drawn up and pushed down alternately.

Frey attributed the spasm to tension of the muscles, and showed that, in his case, the reflex action did not take its origin in the skin; but from one of Bamberger's cases, on the other hand, it would seem as if the latter mode of origin were possible.

Diagnosis.—The jumping may suggest spasmodic tic, or one of the allied conditions that have been described in that section; but apart from the fact that in saltatory spasm no muscular contractions occur except when pressure is made on the feet, the lower limbs are the parts most characteristically affected, and there is no tendency to explosive utterances or mimicry, nor to fixed ideas—features which are so prominent in the clinical picture of convulsive tic. There is little likelihood of confounding the condition with chorea, for, though the involuntary movements of the legs in that affection may be exaggerated by walking, they are usually also well marked when the patient is recumbent; moreover, they are wanting in the regularity and precision of those of saltatoric spasm, and are increased when the subject is under observation.

Treatment does not appear, as a rule, to influence the course of the malady; and, though morphia seemed to have a beneficial influence in one of Bamberger's cases, sedatives appear useless. In one of Sir William Gowers' cases the manifestations of the affection were dissipated, on the second day of attack, by means of free diaphoresis in a hot air bath. The line of treatment on which most reliance must be placed is that of administering tonics, and adopting other measures calculated to build up the system generally and to improve the tone of the nervous system.

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HEAD-NODDING

SYN.—*Head jerking; Nodding spasm; Spasmus nutans; Salaam spasm; Gyrospasm of the head; Eclampsia rotans.*

Introduction.—This is an affection of early infancy in which nodding, lateral or rotatory movements of the head occur from time to time, with or without nystagmus.

Of the names that have been proposed for the condition, "Head-nodding," imperfect as it is, is on the whole the most satisfactory; some of the others suggest a spasmodic character, whereas the movements are peculiarly free from spasm; and others again are apt to lead to confusion with a somewhat different affection—known variously as "epilepsia nutans," "eclampsia nutans," and "tic salaam,"—which is a form of epilepsy.

The first published observations said to be on head-nodding were by Romberg and Henoch in 1851. In 1890 the late Dr. Hadden published a paper on the subject in the *Lancet*, and subsequently another account, based on a larger number of observations, in the St. Thomas's Hospital Reports for the same year. Two years later Dr. Peterson of New York recorded his experience of five cases of the affection in a paper entitled "Gyrospasm of the Head in Infants." Among those who early called attention to the condition in this country are Dr. Stephen Mackenzie and Dr. Gee.

Causation.—No special hereditary influences appear to have been traced in the large majority of these cases; but in exceptional instances more than one member of the same family has been affected. M'Gillivray has, however, recorded a remarkable series of cases in which nystagmus and head movements were met with in adults in whom the condition was congenital and could be traced with certainty through four generations, and there is a probability that the manifestations of the malady had appeared two generations earlier still. The condition is essentially one of early infancy, most cases occurring under one year of age, the majority between six and twelve months; though Henoch has recorded a case in a child as old as three years. In M'Gillivray's cases the patients reached adult life with the malady still upon them; and Bannatyne has recorded a case, in a man aged forty-three years, in whom the affection dated from birth or soon after. The age position in a family seems to make no difference, and, according to Hadden's figures, males and females appear equally liable, though Aldrich alleges a preponderance of females, a state of things which obtained in Raudnitz's series of cases.

Henoch attaches especial importance to dentition as an immediate cause, although he suggests that other forms of reflex irritation may be similarly operative. Hadden, however, met with cases in which dentition neither caused nor aggravated the movements; and in most of his cases the usual local and general signs of disturbed dentition were absent. Moreover, head-nodding may make its appearance at a time when teething is not active, as for instance at the age of two months, and even younger. Peterson's observations also fail to support Henoch's view. It is, however, worthy of note that relapses have occurred in cutting of a tooth, and that in cases of head-nodding teething is occasionally late and slow, even in the absence of rickets.

The children are usually in excellent health, and there appears to be no reason to regard artificial feeding as a factor in causation. Although only two cases in Hadden's first series presented signs of rickets, this condition was evident in nine out of the twenty-one cases of his second series. Congenital syphilis appears to play no part in the etiology. Any debilitating disease may aggravate the movements; whooping-cough, for instance, measles, and aphthous stomatitis have played this secondary part.

The patients have, as a rule, been born in natural labour; but after birth a history of falls and blows to the head has been obtained often enough to justify the belief that such traumatic influences may play a part in the causation; indeed, Peterson, Hirsch, and others regard traumatism as the most potent of the proximate causes. Peterson reminds us of the effect of a severe concussion of the brain in adults in evoking nystagmus, although this is only temporary. Some of the relapses that have occurred after the movements had ceased have certainly been closely related to injuries.

There does not appear to be any close relation between this affection and the convulsive state in general. In only three of Hadden's second series of cases was there a personal history of convulsions, although in six instances other children in the family had had convulsions.

Some of the children have had a peculiar way of looking at objects; but most careful testing of vision in these cases has failed to discover any defect, and the ophthalmoscopic appearances have been normal.

Pathology.—As none of the cases of head-nodding appear to have furnished an opportunity of necropsy, we know nothing of its morbid anatomy. It is highly improbable, however, did the opportunity for examination present itself, that any gross changes would be found in the nervous system to account for the affection. The malady obviously belongs to the large group of functional neuroses in which no morbid histological change is to be detected by the most delicate methods of examination that we have as yet at our command. Consequently, as with the other members of this great group, its pathology is uncertain and obscure.

Of the different surmises concerning its nature that of Hadden has most to recommend it; he regarded the symptoms as due to instability of the motor centres of the cerebral cortex with consequent disarrangement of movements during their evolution in the infant. He points with much reason to the fact that the first acquired movements of the young child are those of the head and eyeballs, which do not become harmoniously associated until the fourth month; indeed, for a considerable time after this date the voluntary movements of the head are ataxic. If, moreover, evolution in some of these children is unusually rapid, as seems to be the case, there is still greater reason why the movements of the head and eyes, being precocious, should be specially liable to disarrangement.

In short, the voluntary or purposive movements of the head and eyes are not at that age sufficiently registered in the psycho-motor centres of the cortex, and dissolution results from the inability of the cortical centres to cope with work which has been too early expected of them.

Others have regarded the condition as more of the nature of a habit or trick, and thus allied to the simple tics or habit-spasms of later childhood.

Symptoms.—Two symptoms are pre-eminent in the clinical picture of this affection; spontaneous movements of the head and nystagmus. This statement is in accord with my own experience, and is further supported by most of the published records of such cases; though at variance with the experience of Peterson, who concluded that nystagmus is not usually associated with the head movements, but is only an occasional concomitant. Despite Peterson's view, however, there can be little doubt that the condition which permits the nystagmus is an integral part of the morbid state which permits the head-nodding.

Although the affection is spoken of as "head-nodding," as a matter of fact a pure nodding movement, such as is made in giving assent, is rare. When it occurs it exactly resembles the movements of the head of a mandarin doll. In one of Henoch's cases the whole of the body was bent forward as well as the head, so that the movement more closely resembled a salaam; but it seems possible that this was a case of eclampsia nutans rather than one of the class we are now considering.

A lateral movement of the head, such as that which signifies negation, is the most common. Hadden evidently distinguished simple lateral movements of the head from rotatory movements; but this distinction is not obvious in the writings of others, and in the cases which I have myself seen the lateral movement was one of rotation. In all of the five cases observed by Peterson the movement was simply rotatory, even without any nodding.

The nodding or lateral movements may be present alone; but more commonly they are variously combined in the same case; thus the one movement may be observed at one time while the other is seen at another; they may alternate during the same period of the affection, at one moment the one, at the next the other manifesting itself; or the one movement may run so rapidly into the other as to make it difficult to say which is the dominant factor in the combined movement which results. Henoch found that in many cases the rotation movements were most marked and the nodding only slight; though he met with nodding more frequently than did Hadden, who only met with pure nodding in a single instance, and in this case the child's mother said that lateral movements were present sometimes.

The movements of the head vary in rate in different cases; they are usually slow, though in some instances they are much more rapid; they may be almost constant, or may occur at varying intervals. Nearly all observers are agreed that the movements are especially prone to occur during efforts at fixation and during excitement; Peterson, on the contrary, stated that they became arrested when the child's attention was called to an object. The movements always cease when the child lies down, when it is asleep, and, according to Mills, when its eyes are covered.

As I have already said, nystagmus is usually present, and, like the head movements, it may be almost continuous, or may occur from time to time at varying intervals. It may be vertical, horizontal, or rotatory; and, although usually present in both, one eye may be affected alone; even when present in both, it is not uncommon for it to be more pronounced in the one eye than in the other; moreover, the character of the nystagmus may be different in the two eyes.

The nystagmus may come on simultaneously with the head movements, may precede them, or may follow them. Now, as head movements may occur alone throughout the whole course of the affection, may there not be cases essentially of the same nature which manifest themselves only by nystagmus—albinism and obvious defects of vision being of course excluded? In my opinion there is much to be said for this view, and little against it.

When nystagmus and head movements are associated at the same period of the affection, they may occur simultaneously, or the two conditions may alternate, so that at one moment head movements are seen, and at the next they are replaced by nystagmus. The nystagmus is much more rapid than the head movements, and has an independent rhythm even when the two phenomena occur simultaneously. The usual rate of nystagmus is about four to six movements per second. It cannot be said that any one variety of nystagmus is constantly related to any given movement of the head (as, for instance, vertical with nodding, and horizontal with lateral movements); for, although in some cases such a relation obtains, in others every variety of combinations is met with. When the nystagmus is unilateral the head may move either to that side or away from it.

The nystagmus, when absent, may be evoked by any attempts at fixation, and also by forcibly restraining the head movements; so, too, either of these devices will accentuate nystagmus already present. It has been noted further that the nystagmus may be more pronounced in certain positions of the eyes than in others; thus in one of Hadden's cases it was most marked when the eyes were directed to one side, and least so when they were turned in the opposite direction; in another child the nystagmus was especially evident when the eyes were directed upwards. Moreover, the character of the nystagmus may change with alteration of the position of the eyes, as in Gordon Norrie's case, in which a horizontal nystagmus tended to become vertical when the eyes were turned upwards. The eyelids shared in the movements of the globes in one of Hadden's cases.

Another ocular defect that has been met with in some cases of head-nodding is strabismus, which appears usually to be convergent. Instances of this association have been reported by Henoch, Gee, Hadden, and Peterson; and slight transient squint was noted in a case recently recorded by Aldrich; Bannatyne's case in the adult also presented this phenomenon. In one of Hadden's cases, in which nystagmus was absent, the movements of the head were preceded by lateral deviation of the eyes.

The pupils usually present no abnormality, though the condition of hippus was present in Norrie's case and in one of Hadden's. The latter observer also found the reaction of the pupils on convergence poor in one case, and in another, in which nystagmus was limited to one eye, the pupil on that side acted better consensually than on direct stimulation. In all the cases examined the discs have been found normal; in two of Hadden's patients the fundus was light coloured; in one of these there was a slight crescent of atrophy round the disc, in the other there was a deficiency of retinal pigment.

I have said that there has been reason to suspect faulty vision in some cases, though careful testing of sight has failed to estimate any defect of the kind. One of Hadden's patients threw its head back, and partially closed its eyes on looking at anything; another of the children looked at objects sideways, and usually to the right; in a third the mother believed that the child could not see with the left eye, because, when its attention was directed to anything, it turned its head to the left and "looked out of its right eye." Yet in none of these cases was Mr. Marcus Gunn able to make out any defect of sight, and the ophthalmoscopic appearances were normal in all of them; it is worthy of note, however, that in the third child referred to above nystagmus was limited to the left eye, and may have accounted for the child's preferring to make use of the right eye alone in looking at an object. Indeed it seems probable that some form of visual defect, or possibly nystagmus, must account for the children's behaviour in these cases.

Attacks of momentary loss of consciousness occur in some cases of head-nodding; the head and eyes may be turned to one side, but without anything of the nature of a general convulsion, the seizure resembling rather attacks of petit mal. In none of these cases has a personal or family history of convulsions been obtained, and none of them has been known to fall into genuine epilepsy later in life.

Mentally the children are usually particularly bright, in some instances even precocious, taking notice unusually early; on the other hand, in a few exceptional cases, there has been distinct mental defect.

In the latter case, it has usually been that the child, intelligent and happy when it first came under observation for the head movements, subsequently became cross and passionate, or less intelligent, irritable, and forgetful; in one of Hadden's cases, a child whose intelligence was above the average had occasional outbursts of laughter. But so far as can be ascertained, even in cases of this kind, no permanent mental defect results.

One other symptom, present in two of Hadden's cases, must be referred to, namely, rigidity in the muscles at the back of the neck; a fleeting phenomenon, like the other manifestations of the affection. In one of the cases the head was retracted, and when passive attempts were made to draw it forward the child cried.

Prognosis is highly satisfactory; in time total cessation both of the head movement and of the nystagmus has been noted in the majority of the cases, though the latter usually persists longer than the former. The duration, however, varies in different cases, and whereas, in some cases, all movements may cease in the course of a few weeks, in others they continue for months. Moreover, after they have ceased, they tend to recur under any exciting or debilitating influence; or the nystagmus may recur without the head movements. In one of Hadden's cases nystagmus was still present after a period of seventeen months, and we have seen reason to suppose that the condition may persist to an advanced age, as evidenced by M'Gillivray's and Bannatyne's cases.

Treatment.—It is probable that without treatment most of these cases would end in recovery in the course of a few months, or it might be of years; but they certainly appear to be favourably influenced by treatment. The drug which appears to be of most service is bromide of potassium, given in doses of one and a half to three grains, three times a day, to infants at the age when the affection is most commonly met with. It is especially important that bromides should be administered in the cases in which attacks of loss of consciousness occur; moreover, such treatment should be continued for a longer time than would otherwise be necessary. Belladonna may be combined with the bromide, and later steel wine or syrup of the phosphate of iron may be substituted for sedatives. The latter measures are especially indicated in the exceptional cases in which nutrition is poor, or there are any evidences of rickets, when cod-liver oil or some preparation of malt will also be found useful.

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ECLAMPSIA NUTANS

Syn.—*Epilepsia nutans; Salaam convulsions, Spasmus nutans; Tic salaam.*

Newnham furnished the first satisfactorily recorded account of cases of this kind in 1849; a year later Willshire published a case, together with an account of the salient features of the affection. In this same year two cases were recorded in Germany by Faber, and, according to him, Romberg would seem to have been the only German observer who had previously noticed it.

The malady is one of childhood, nearly all cases occurring between the period of first dentition and puberty; it is characterised by peculiar bowing movements of the body, the whole trunk moving forward and downward, so that sometimes the head nearly touches the knees; in other cases, however, the body and head are only bent slightly forward. This movement occurs in paroxysms which may be repeated as often as 50 to 140 times a day; and each attack may be made up of 20 to 100 bowings. In other instances the attack has occurred once a day only, and that at some stated hour, as for instance, in one of Faber's cases, at 11 a.m.; a second attack sometimes occurred in the evening. Sometimes the patients are free from attacks for intervals of a few days. Nodding movements of the head accompany these "Salaam convulsions," as they have been called; moreover, nodding movements of the head may be the first abnormal phenomenon, the bowing forward of the trunk being added subsequently, as in the case of his own child recorded by West in 1841.

The severer bowing attacks have been observed to follow sleep; and it is said that their severity appears to be sometimes proportional to the depth of the preceding sleep. During sleep, however, all such movements are in complete abeyance. During the attack the child often seems bewildered, but consciousness is not completely lost; and, though after a paroxysm the patients may be drowsy and exhausted, in other cases they are quite bright and happy as soon as the bowings have ceased.

As the case progresses more general automatic movements are observed to occur, and these may take the form of epileptiform or tetanoid attacks; and subsequently paralysis, of paraplegic or hemiplegic type, has been met with in some instances; though this does not appear to be an essential part of the malady. In regard to the epileptiform attacks momentary loss of consciousness has been observed, in some instances, immediately preceding the convulsion in the limbs; in other cases of eclampsia nutans no affection of consciousness has been noted. The epileptiform attacks may increase in frequency, and indeed the child may ultimately become epileptic.

There is a pronounced alteration of the mental faculties in these patients, so that, although apparently perfectly normal in intelligence before the onset of the malady, they subsequently have an imbecile appearance, and their mental faculties do not give evidence of having reached a due stage of development. Finally, complete imbecility or idiocy may result, from which the patient may, however, recover, though slowly and with difficulty.

This affection does not appear to have attracted much attention during recent years; but many of those who have furnished us with accounts of the condition already described as "head-nodding" have insisted that that affection is to be distinguished from eclampsia nutans, which latter condition they assert to be an independent malady, and, moreover, an epileptic manifestation.

To deal with the second point first: it is open to question whether the assertion that eclampsia nutans is a form of epilepsy is justified by the facts at our disposal. Faber was of opinion that the malady could not be so regarded, seeing that loss of consciousness, one of the principal symptoms of an epileptic attack, is sometimes absent, as in one of his own cases. That true epileptic attacks may occur in association with the other manifestations of the affection does not prove that all the manifestations are epileptic. We might indeed, with as much show of reason, regard head-nodding itself as an epileptic condition, as attacks of petit mal are sometimes associated with the other phenomena; so again paramyoclonus should be similarly regarded, for of it epilepsy is no uncommon accompaniment.

In considering wherein the malady differs from that which has already been described under the title of head-nodding (p. 903) there are several points deserving of attention. Although cases of eclampsia nutans have been met with in families free from any hereditary taint of neurosis or mental affection, in other instances the converse has been notable. Then again the condition manifests itself later in childhood than head-nodding, so that, although it may appear at six months, it may not do so until the child is as many years of age. So, too, the more general automatic movements prone to occur with "salaam convulsions" are foreign to the ordinary cases of head-nodding. Moreover, the association of eclampsia nutans with epilepsy appears to be more intimate; and while the attacks of petit mal which may accompany head-nodding do not persist, the attacks in eclampsia nutans may, as I have said, continue permanently as genuine epilepsy. Again, although some mental defect has been observed in certain cases of head-nodding, it has never attained the degree met with in eclampsia nutans; furthermore the mental defects have been transitory.

That no very hard and fast line separates the two affections—head-nodding and eclampsia nutans—must be evident from what has already been said of the maladies. To emphasise this point further, however, it is worthy of note that, in one of the cases included by Henoch in his description of head-nodding the movements were not confined to the head, but the whole body bent forward, as in eclampsia nutans; Finlayson, on the other hand, in 1877, recorded a case as one of eclampsia nutans which unquestionably should belong to the category of head-nodding, if a distinction between these two affections is to be drawn. Nystagmus was present in this latter case, as in others recorded as instances of eclampsia nutans; but how many of these cases were head-nodding proper it is not easy to estimate. Squints have also been noticed in cases of eclampsia nutans.

Eclampsia nutans may then after all be but a more aggravated form of the same malady whose milder manifestations are seen in the condition known as head-nodding; and even if the evidence be considered insufficient to allow of our coming to so definite a conclusion, it must be admitted, nevertheless, that the two affections are closely allied.

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no support to the existence of any special danger at "the change of life," the importance of which transition has certainly been very much exaggerated.

Age, sex, and heredity.—The influence of heredity is considerable at every age, although it lessens progressively as life goes on. Combining the cases seen in hospital work with those seen in private, we find the total percentage of heredity is 41 per cent—about 8 per cent less than that found in the cases seen only in private. In the first 20 years the percentage of heredity is 44, in the second 39, in the third 29. Cases with inheritance are met with up to the latest period of life at which epilepsy begins.

The proportion of the sexes in the cases in which heredity can be traced is almost the same throughout life as in the total number at each period of life.

The large proportion of cases in which no predisposition can be traced need occasion no surprise, if we remember that the conditions for the sudden release of nerve energy essential to the normal action of the brain, must entail a liability to abnormal readiness to discharge on such slight disturbance of nutrition as may be conceivably brought about by causes which we cannot at present recognise.

Proximate causes.—So far as we know and can interpret the facts of the disease, we regard a certain persistent condition of the nerve tissues as the most general element in the causation. In a considerable proportion of the cases, however, the first attack is referred, with more or less reason, to some more immediate cause. In most cases this morbid influence is too slight to do more than determine an event for which the conditions exist. It is either like the gradual rise of temperature which brings about explosive combustion, or a spark which immediately excites a discharge of explosive material.

In one-half the cases a proximate cause can be traced. Such a cause is more frequently met with in males than in females; perhaps because the predisposition is more powerful in the latter and needs an exciting influence less frequently. In infancy, nevertheless, the first fit seems to be excited as frequently in one sex as in the other. After puberty, and to the end of life, a proximate cause is recognised more frequently in males; and after 50 it seems to be rare for the onset not to be distinctly excited.

Particular causes.—Infantile causes.—A considerable number of cases of epilepsy date from infantile convulsions. In most of these the first fits occur during retarded dentition (commonly with other indications of the state called rickets) as definite convulsions, or as very slight "minor" attacks; these persist to puberty, and then become definitely epileptiform. In other cases they may cease for a year or two and recur as minor attacks. There is every gradation between persistency and an interval lasting from the first dentition to puberty. This suggests that the early convulsions had produced a lasting modification of the brain, one facilitating the tendency to discharge at certain developmental epochs, and when some excitation induced an attack. In about one-tenth of the total number of cases of epilepsy some such influence must be ascribed to infantile convulsions.

The popular connection of these fits with the process of teething has probably a very slight foundation in fact. A slight local irritation may have an exciting influence, but the chief factor is the general retardation of development. This of necessity has most influence on the nerve structures, which are either the least developed structures, or have most recently attained structural completion. The highest, controlling centres are latest to develop, and will suffer most from any hindrance to development. The motor centres which subserve reflex processes in the cord and brain have then an insubordinate excess of function; and among these lower centres we must include the structures of the motor cortex as well as those of the spinal cord and mid-brain. It is not difficult, therefore, to perceive a process by which simple retardation of development shall induce the reflex over-action which gives rise to tetany, to carpo-pedal contractions, to laryngismus stridulus, and to actual convulsions. The influence which is exerted by this process leads to ordinary idiopathic epilepsy; but the attacks which connect the early and later attacks are often those of the minor form, and their character and significance is commonly disregarded by those who have the care of a child so afflicted.

In connection with the early origin of epilepsy, it should be noted that cases of another class are due to the sudden occurrence of a cortical lesion on the surface of the brain at some time in the first two years of life. This may cause hemiplegia, transient or persistent; or an initial weakness so slight as to attract no notice, especially if the lesion be the result of some prostrating illness. In the latter case the lesion is near the motor centres, but does not involve them; and disease so situated is especially apt to give rise to subsequent convulsions. These cases are distinguished by the severity of the initial convulsions, by their frequently unilateral character, and by the fact that the slighter fits at a later age distinctly begin on one side and involve this chiefly. Although such cases are essentially different from those of idiopathic nature, the frequently recurring discharges seem to induce a similar state of the brain; and not uncommonly minor attacks appear quite similar to those of the idiopathic form.

In a few cases, which must be referred to infancy, although the attacks may present themselves at a later period, the cause is damage to the cortex during the process of birth. The amount of mischief in these cases is seldom sufficient to cause the characteristic bilateral motor affection known as "birth palsy"; and the recognition of the cause is often difficult. Cortical injury is to be suspected, however, when convulsions are manifested in the early infancy, or after dentition, of first children whose birth had been tedious; and this is especially probable when symptoms during the first days of life point to some amount of meningeal hemorrhage.

Injury in or near the motor cortex may lead to recurring convulsions; but these begin in particular parts which correspond to the position of the lesion. These cases are rare. In some cases, however, general convulsions, like those of idiopathic epilepsy, follow some more general concussion which has possibly a widespread influence on nutrition. Yet it must be remembered that injury involves a mental as well as a physical shock, and mental shock, as will be seen, is a potent cause of epilepsy.

Toxic influence.—A first fit may occur during or immediately after an acute specific disease; but only one specific disease has this sequence in any considerable proportion of cases of epilepsy. This is scarlet fever. The first fit may occur during the disease, and when it occurs after it there is seldom any renal sequela with which it can be associated.

In rare cases some other toxic state of the blood is the apparent cause. This may be due to some perverted chemical process within the body, such as that which results from chronic kidney disease, in which recurring convulsions may apparently arise independently of uræmia in the strict sense of this word. It must be remembered that the abnormal chemical process which we can trace must be associated with other chemical changes that we cannot trace, and that these may often have far more toxic results.

Emotional disturbance.—As a direct excitant of the first fit intense sudden alarm takes the first place. It can be traced in a considerable proportion of cases. In many instances the cause of the alarm seems absurdly inadequate; but that which seems inadequate at first sight may not seem so on further consideration. An illustration may be given to which particular exception was taken by a French author, when it was first mentioned, on account of its trivial character. Mere minuteness of excitant, however, becomes unimportant if it act as the spark in an explosion of gunpowder; and it must be always kept in mind that the essential cause is the internal predisposition. The instance referred to is that of a nervous sentinel who was on duty one dark night near a churchyard. A white goat suddenly ran across the churchyard and jumped upon the low wall. The man, convinced it was a ghost, shook with terror, but was unable to desert his post. An epileptic fit soon followed, which was succeeded by others.

The disturbing effect of sudden fear seems the greater when it cannot have its normal consequence, namely, the energetic discharge of the motor centres to escape from danger. There is action upon them, but it is perverted in effect; it disturbs their nutrition and deranges their function. The immediate effect is seen in the trembling, which has thus become a synonym for fear; the remote effect is seen in such maladies as epilepsy, chorea, paralysis agitans.

A true epileptic fit seldom follows the sudden fright instantly. An instant fit is more commonly hysterical, and sometimes perhaps preserves the patient from the more enduring malady. An epileptic fit generally occurs within a week, and if a longer interval elapses the relation to the cause is doubtful.

Prolonged anxiety seems also to be a definite cause, although the more gradual mode of its influence makes it less easy to trace with certainty. Acting upon a predisposed person it seems to be influential at all ages, and in the later period of life often without any predisposition. The affection commences then chiefly in the male sex, and it is rare for the sufferer not to have been subjected to a long mental strain. The way in which this cause acts is less perceptible than in the case of sudden fear. It doubtless depresses the nutrition of the whole nervous system, for there are few chronic degenerative diseases of which it may not be a cause; but probably its influence in producing epilepsy, as with the other causes in earlier life, is the result of a definite predisposition on the part of the structure concerned; how this predisposition arises we have no knowledge.

Reflex causes.—The chief peripheral irritation which can be regarded as a cause is that of intestinal worms. It seems to be only from irritation in the intestine that general convulsions result. These cases illustrate the manner in which convulsions are self-perpetuating by the primary disposition already mentioned.

Irritation of the nerves of the limbs is so rare a cause of epilepsy that doubt as to the occurrence of fits from this cause is not unreasonable. It is certain that they occur too seldom to merit special description.

Symptoms.—The severe epileptic fit is that which has always been the recognised type of the disorder, and therefore may be first described. The sufferer, with or without some momentary sensation—a sensation at the epigastrium, or of some special sense of vertigo or the like—becomes unconscious and falls, often with violence, as if hurled down. The fall is often such as to cause some injury to the patient, or to involve him in some danger, as of fire or water. The fall is due to involuntary spasm inconsistent with the maintenance of the upright posture, whether of standing or sitting. The spasm is tonic, and affects all the muscles of the body; in those of the chest it occasions an expiration which, if it coincide with spasm of the larynx, may cause a strange cry.

Although general, the spasm is seldom equal on the two sides; the head therefore deviates to the side on which the spasm is most intense, and the arms are not moved equally. They are often slightly flexed at the elbow, which is separated from the trunk; and the fingers are in strong "interosseal flexion." Occasionally the hands and wrists are strongly flexed, in which case sometimes the elbow is also flexed and the hands are brought in front of the body. The legs are usually extended, although often not completely. In some cases, with strong flexion of the arms, the legs are drawn up. The mouth generally deviates a little towards the side of the greater spasm, towards which also the eyes are often directed. After about 30 seconds, during which time the arrest of breathing renders the face cyanotic, tremulous variation in the tonic spasm is observed; this soon increases to definite remissions, and to the jerks of the clonic stage. The remissions deepen until they become intermissions, and soon afterwards, at the end of about another minute, occurs the last jerk, which is often as violent as those which have preceded it; the patient then lies in a state of exhausted relaxation. During the stage of clonic spasm the cyanosis lessens, because there is movement of air into and out of the chest; this causes saliva to be frothed out of the mouth, often attended with blood in consequence of the tongue being bitten. The tongue shares the clonic spasm, and on account of the inequality of this the tongue is pushed to one side, so that the edge is cut between the jaws. As it is very rarely pushed between the teeth before the jaws are brought together, the tongue is not usually bitten in the stage of tonic spasm. Some patients never bite the tongue, apparently because of some special character of the spasm. Urine is often passed during the attack, a symptom which is probably due to extension of convulsive spasm to the wall of the bladder, seeing that it is discharged with violence, and in no proportion to the degree of general muscular contraction.

Attacks occur of less severity in every degree, even to that in which the occurrence of muscular contraction can scarcely be recognised. In idiopathic epilepsy the initial tonic spasm is a general characteristic, and in the slight fits it may be the only spasm. A brief stillness may indeed alone represent an attack. Cases in which a general convulsion of very slight degree consists only of tonic spasm are exceedingly rare.

In this respect convulsions which begin locally, from local disease of the brain, stationary or active, present an absolute contrast to the idiopathic form. In the former the attacks begin by local clonic spasm in the part related to the spot in the brain at which the disease has raised the excitability to a morbid degree. The spasm thence spreads throughout the side, and then may involve the other side. If the discharge is intense the spasm may extend with such rapidity that the second side seems to be involved almost from the outset. Moreover, the first clonic convulsion may quickly change to tonic spasm, which then runs a course similar to that presented by the idiopathic form. This change is apparently promoted by the effect of repeated discharges on the whole motor gray matter of the brain, which produces a tendency to general discharge that may ultimately be almost as intense as in idiopathic epilepsy, and must be considered in assigning such cases to their proper position. This view is emphasised by the fact that the minor attacks, presently to be described as occurring in the idiopathic form, may appear in the cases of old stationary organic disease.

In such severe fits consciousness is not always lost at the onset. In a small proportion of them some abnormal state or symptom exists for an hour or two before the attack. In a much larger proportion the onset of the attack is preceded, for a second or so, by some sensation or other disturbance of consciousness. This is called the "warning" of the attack, or, in medical terminology, the "aura." This old Latin name for the premonition seems to have arisen from special notice of the cases in which local disease of the brain and local discharge cause such partial commencements of the attack as twitching contractions, the clonic spasm above mentioned, or some sensation in the part, generally tingling, which begins in an extremity and passes up a limb. But it is probable that the origin of the term was connected with the idea, dominant then and for many a century afterwards, that the arteries contain air; and the ascent of the sensation was attributed to the ascent of an unnatural vapour in these vessels. The term "aura," like "hysteria" and many others, has survived its original associations, and persists as a simple synonym for the immediate warning of the fit.

This immediate warning is of great importance. Except in the cases of extreme local instability of the cortex, generally due to local disease, the warning is some sensation. The sensation is the effect on consciousness of the initiating process of discharge, which begins in structures through which those are influenced that are highest in function, related, that is, to consciousness. The warning is thus an indication of the part of the brain in which discharge begins, because the place where it first attains such an intensity as to cause a "sensation" must be assumed to be the place at which it starts. In general the sensation is uniform in the same case; occasionally it undergoes a change in the course of time.

The importance of these warnings is increased by the fact that they are perceived more regularly and in greater degree the slighter the attack. This is true through the whole range of degrees, until at last the lowest level is reached in which attacks may consist only of the "warning" or "sensation"; sometimes with imperfect loss of consciousness, sometimes with no appreciable impairment of it. This has led to the popular use of the word "sensation" as a synonym for the minor attacks. As a rule the warning of the slighter attack, with definite loss of consciousness, is the same as that of the severer forms, should consciousness persist long enough for perception. Sometimes, indeed, the slighter attacks are heralded by a different warning, but the common correspondence makes it well to consider the warnings of attacks of all degrees together.

The relative amount of the process of discharge that is perceived by consciousness varies. As already intimated, it may be the whole, the disturbance ceasing with the warning, consciousness not being impaired. In other cases there is merely a partial impairment of consciousness, enough to prevent perfect recognition of the surroundings, so that they seem to be unfamiliar and strange. Every form of aura may, however, be followed by definite loss of consciousness, which in slight attacks is usually momentary. It is prolonged in many cases, in appearance, and in reality so far as the self-consciousness which involves memory is concerned. There may be a moment in which the patient is obviously unconscious; this is succeeded by a few minutes of automatic action, rambling talk, attempt to undress, and the like, in which another person would not imply unconsciousness; although the patient may remember absolutely nothing of that which has occurred. Such a state may follow an attack in which there was slight spasm, but it hardly ever follows an attack with severe convulsion. This sequel will be considered presently.

Prodroma.—Besides the immediate warning, or when there is none, a few patients are conscious of some unusual symptom which tells them that an attack is impending. There may be peculiar mental irritability, or sudden jerks, or definite minor seizures. It is important to keep these distinct from the immediate warning, since a patient when asked if he or she knows that an attack is coming on, will often say "Yes," and describe such prodroma, even when there is no immediate warning.

Forms of aura.—The chief forms of warning may be briefly considered. Their interest is great, but their practical value is almost limited to the detection of attacks, which might otherwise be unperceived, by the evidence this character affords, and by their indication that the instability is the result of a local lesion of the brain.

Motor phenomena.—Local clonic spasm in the limbs is too rare in idiopathic epilepsy to be worth mention, except in connection with organic brain disease.

A definite but unusual commencement is by the act of running. A patient may run forwards several hundred yards, and then fall in a fit. Another may turn round and walk back, and then fall. Another may turn round two or three times, and then fall unconscious in an epileptic convulsion. We cannot really explain these co-ordinated motor symptoms, but the last is obviously connected with the most common warning of which a memory is retained by consciousness; namely, vertigo.

The giddiness which is a common warning of an epileptic fit seems to be a purely motor symptom, the result of the influence on consciousness of the greater energy of the centres of one hemisphere. Hence as a rule the patient seems to turn towards the side which is most convulsed; and it seems to him that other things are turning in that direction, because he imputes to objects the movement he seems to have. If this terse description seems not to be clear, it will probably become clear if carefully thought over. Occasionally the features of the vertigo described are difficult of explanation.

Vertigo involves a false perception of the relation of the individual to his environment, and this constitutes inaccurate, that is, imperfect consciousness.

But in simple vertigo, apart from epilepsy, definite loss of consciousness is almost unknown. In epilepsy it is almost, perhaps quite constant. The sensation of turning may be associated with a visual aura, the appearance of an object which moves from one side of the field of vision to the other, and which the patient seems compelled to follow until its disappearance. Often the side on which the object appears is that chiefly convulsed, the side of the body, that is, opposite to that of the brain in which the chief discharge takes place; and the disappearance of the object on the opposite edge of the field of vision is followed by a turning of the head in the convulsion. This fact illustrates the extreme perplexity of the features of the vertigo in the epileptic discharge, and indeed alike of vertigo and of the epileptic warning in general. Those who desire to pursue the subject further will find some information about it in my Bowman lecture (2). The leading fact, however, is the evidence of the motor character of the warning, associated as it is with deviation of the head and sometimes with rotation of the body. Moreover, as sudden vertigo is often the only subjective indication of a minor attack in which there is a moment of absolute unconsciousness, this symptom is of great practical importance.

Local onset.—In idiopathic epilepsy there is seldom more definite evidence that the discharge occurs earlier in one hemisphere, than is afforded by the evidence of excess on one side which is shown by deviation of the head; but in other cases the onset may be indicated by a sensation in the extremity of one limb only, usually by a tingling which passes up the limb; or there may be clonic spasm associated with the sensation, or occurring alone. The sensation or spasm may begin in some part of the hand, for instance in the thumb and finger. There may be a similar beginning in the foot, or in some part of the face, generally the angle of the mouth. The spasm may remain local, or may spread through the side, or to the other side, with varying rapidity. In all such cases there is excessive local instability in the cortex of the brain, in the centre related to the special symptom. Such excess of local instability is very seldom part of idiopathic epilepsy. It is commonly the result of old local organic lesion, which has damaged the gray matter, and made that which is least damaged functionally unstable. An infantile lesion is the most common cause, but they may be due occasionally to thrombosis or embolism in later childhood or early adult life. The gray matter of the brain is normally so sensitive that discharge spreads rapidly and widely in proportion to its intensity. Repeated discharges establish a widespread habit which is practically identical with the general brain state in idiopathic epilepsy. The local disease which causes the instability is often such as to cause some degree of hemiplegia, especially at the onset. Persistent hemiplegia is usually due to destruction too extensive to permit discharges; hence these cases are often called "post-hemiplegic epilepsy." It is important, however, to note the manner in which the tendency to discharge becomes established. After removal of a small superficial scar attacks have continued; moreover, the convulsions which begin locally, in the hand for instance, and spread thence, may be accompanied, after a time, by minor attacks, quite like those of idiopathic epilepsy. Such facts prevent an absolute withdrawal of cases due to old local disease from those which are due to some general state of nutrition or functional proclivity. We can separate the cases of local disease, sometimes included under the general designation "post-hemiplegic epilepsy," by their causation; but we cannot always, or indeed often, separate them by their course or ultimate features.

One fact regarding this warning deserves note. It may be purely sensory, apparently a gentle discharge in the sensory elements of the central region. This aura may be the result of organic disease, and is one of the facts which show that the central region, whence the motor impulses proceed, has also sensory functions. A gentle discharge of this kind, causing, for instance, a sensation of tingling in the foot, may pass up the leg, up the side of the body, down the arm to the hand, and when it reaches the fingers clonic spasm may occur, which passes up the arm; at this point, as a rule, consciousness is lost in a severe attack. It is as if the discharge in the sensory elements of the centres rippled through the sensory layers until it reached a structural and functional limit, and was there turned aside to the motor structures, in which it quickly gathered force. Again, such a sensory discharge—in the arm, for instance—may be accompanied by sudden powerlessness of the arm without spasm. Apparently the discharge in the sensory elements simply inhibits the motor structures. If a motor discharge begins in the centre for the foot and causes clonic spasm, this, passing up the leg and side, may begin again in the hand on reaching that level; it does not descend the arm from the shoulder.

Bilateral and trunk auræ.—Occasionally the warning is some sensation referred to both legs, which seems to ascend, often to the head; or it may be a sensation in the spine, which is rare and not important. From such trunk warnings it is necessary to separate those which are included in the next class.

Visceral auræ.—The onset of an attack is often announced by some sensation referred to the viscera, the epigastric region, the cardiac region, the throat, or the head. With the exception of the cephalic warning, these visceral auræ may be referred to the region whence impressions reach the centre through the pneumogastric nerve; the chief functions of this nerve are represented in the gastric, cardiac, and throat sensations.

The epigastric aura is one of the most common. It may be a deep-seated pain, sometimes associated with nausea. More often it is a peculiar undescribable sensation, which often ascends to the throat, rarely to the head. When it seems to pass to the head there is immediate loss of consciousness. If it passes up to the throat it becomes there a sense of constriction or fulness, and of breathlessness—being similar, apparently, to the globus hystericus. The pain which remains at the epigastrium as a symptom associated with nausea may reasonably be regarded as due to the central representation of the gastric function of the vagus. This is compatible with another fact, namely, that the sensation is occasionally referred to a lower part of the abdomen, since some fibres of the vagus pass to the intestines. On the other hand, the sensation which ascends to the throat and is associated with a feeling of want of breath, may be referred to the central representation of the respiratory function of the nerve. How extensive this is we can perceive from the relation to respiration of the various ways in which emotion is manifested. In the me class may be included cardiac sensations which are not common d are seldom well defined. The most common of these is palpitation.

Cephalic sensations.—When an epigastric aura ascends to the head, reaching it consciousnesss is usually lost before the character of the phalic sensation can impress itself so as to be revived. This is an teresting fact in relation to the representation in the sensory centres f the brain itself. But occasionally a definite physical sensation in the ead is the immediate warning. It can seldom be described except as "a rushing," or "fulness," or "movement in the head." This lasts presents gradation to vertigo by a feeling as if something were turning round in he head, now and then associated with a sense of movement of external bjects.

Olfactory and gustatory.—Special sense warnings are not uncommon. he order of frequency is visual, auditory, olfactory, gustatory. It is nstructive that flavours, which are perceived only through the olfactory erve (and being caused by substances which enter the nose through the osterior nares, and cause a simultaneous stimulation of the true gustatory erves), seem to be associated with taste in the central discharge which auses the warning. When this is described as a "smell" there is no ensation of a flavour. The difference in the mode of stimulation of the ame nerve seems to be reproduced in the centres. The olfactory and gustatory warnings are almost always unpleasant, but generally transcend the powers of description and even of comparison. Our vocabulary is quite inadequate to the expression of normal sensations, and far more so for those which are due to processes that seem unlike those which are excited in the normal action of the nerves.

Auditory warnings.—These are also uncommon. There is occasionally some simple sound at the moment of onset, such as a whistle, the sound of a bell, or a crash. Occasionally there is a more elaborate sound, such as music, or voices, and even distinct words. The latter are important for the reason to be mentioned in connection with the more elaborate visual warnings with which the similar auditory aura is sometimes associated. Inhibition of the auditory centre may precede the fit; there is sudden silence, which may be associated with sudden darkness, before consciousness is lost. The loss of hearing may precede or accompany the subjective sound, an association of inhibition of response to external stimulation with a spontaneous action on the centres related to consciousness.

Visual warnings are more common than any others connected with the special senses. The extreme delicacy of the structure which responds to the most rapid form of motion of which we have direct perception must be associated with a corresponding delicacy of action in the central structures that receive the nerve impulses produced by the waves of light. The marvellous difference in the influence on consciousness produced by waves which differ only in degree, and not greatly in degree, as for instance in the case of the sensations of red and blue, is a fact of far-reaching significance. It is not surprising that, when central instability exists, the visual centre should often lead the way in the sudden derangement of balance. The warning may be a flash of light, stars, sparks, simple light, or definitely coloured. There may be more complex sensations of objects, faces, persons, scenes; and with the last an auditory aura of corresponding elaboration may be associated. It is important to note that in those patients who experience such psycho-sensory auræ there is a strong tendency to mental derangement.

Inhibition of the visual centre—sudden darkness—is a frequent warning. It may be followed (it is seldom preceded) by a "discharge" in the centre; first all becomes dark, after which stars or a luminous object appear. This is an important indication of the close relation of arrest of action and spontaneous action, of inhibition and discharge.

The various special sense warnings are occasionally associated, most often the auditory and visual. The degree of elaboration in each usually corresponds, although sometimes an aura of low degree is succeeded by one of greater elaboration. Two lights in one case were always succeeded by the figure of a woman. In the case of both the visual and auditory sensations there may be a progressive increase or diminution; lights which suddenly appear may become fainter and seem more distant, or seem to approach; sounds may become slighter or seem to be louder and louder. In each case, with a considerable degree of change, consciousness is lost. No interpretation of these phenomena can be given beyond that which is at once obvious to every one who considers them.

Psychical auræ.—A sudden abnormal mental state may constitute the first symptom, and may leave a definite impression upon the memory. The brain processes which attend mental states may leave a residual effect which permits their subsequent revival; and their perception may be called, somewhat loosely, "psychical sensations." That which precedes an epileptic fit may be an emotion or an idea. The emotion is almost always some form of fear. It may be definite alarm, and associated with the conception of some cause for it, as that of a pursuer from whom the patient has to run away, and does actually run, sometimes looking back in terror. It may be more vague in character although definite in nature, as a sense that whatever is being done is morally wrong.

Those warnings which take the form of "ideas" are generally of the nature of complex special sense conceptions, especially related to vision. Not only may a sense of fear be accompanied by the vision of some cause for it, but the more elaborate visual sensations are such as to involve a psychical process. A remarkable instance is that of a woman who saw London in ruins, the Thames emptied to receive them, and herself the lonely survivor—a manifest psycho-sensory warning. Indeed the same combination may be discerned in many other forms. Its details have no other importance regarding the disease than indication of a special instability of the centres which are concerned with psychical processes.

Somnolence.—These warnings illustrate the many-sided relations of the condition which subserves subjective consciousness, and through which disturbance may cause its apparent arrest. Consciousness may be lost and return, with such suddenness that no trace of either process is retained by the memory, and the patient is absolutely unaware of the gap in his memory. But it may also be lost deliberately, by a gradual process which is described as "sleepiness," or sometimes by a more analogous state with a slight psychical character, called "dreamy." This warning is more common in minor attacks, and especially in those which are slightest in degree and least rapid in evolution. Closely allied to these is a more definite mental state, especially common at the onset of minor attacks, and sometimes apparently constitute them, in which there is a sense of strangeness, a sense of unfamiliarity—it may be with a place in which the patient has spent his life.

Minor attacks.—"Epilepsia minor" is the name applied to the attacks in which there is no visible convulsion. "Petit mal" is used in the same sense, and indeed more generally, because the French taught us to recognise the nature of such attacks, and we have therefore called them by the French name. There is no definite distinction between the two classes; spasm may occur and yet be unseen, and slight momentary visible tonic spasm may easily raise a doubt into which class the attack should be placed. It would indeed be well to recognise an intermediate class, which might be called epilepsia media, in which there is muscular spasm of tonic character, without the clonic spasm which follows when the tonic spasm is more severe. In the epilepsy which is due to organic disease the slight attacks consist of tonic spasm only, but these differ in so many particulars from the slight attacks of the idiopathic disease that it is not practicable to consider the two together.

In the popular nomenclature various names are given to these attacks, according to their chief characteristic. When they begin with a sensory aura, of which only the patient may be conscious, they are called "sensations." If loss of consciousness occur they are called "faints." "Turns" is another name for them, which seems only due to the common use of the word as a designation for any sudden unusual falling. It is always important to ascertain what word is commonly used. It should be a matter of habit never to use the word "fit" or "epilepsy" unless it be known that it has been employed before in the patient's hearing. Friends often conceal the nature of the attacks, and when their nature is not known by the patient it is well to avoid so painful a word.

Varieties.—The forms of minor attacks are so various as to baffle classification and preclude full description. The typical attack is a momentary loss of consciousness, causing a momentary cessation of conversation; often a vacant look, or the dropping of anything from the hand, and occasionally a fall. There is no pallor of the face at the moment, although this often immediately succeeds the attack, which is so brief that a patient is only observed after it. Hence the erroneous idea that pallor is a symptom of the attack itself. Nor is there any change in the pulse at the time, or in the aspect of the retinal arteries, as I have been able to observe. Of such loss of consciousness a patient may be wholly unaware, so sudden is its onset and ending. He may go on with the sentence he is uttering, and be astonished to observe that those about him are looking at him with concern.

Such change in consciousness may be incomplete. It may not be lost, but only rendered for the most part imperfect, and the character of the imperfection varies in different cases. It may be purely mental; the patient sees that which is about him, but it seems strange and unfamiliar. Much less frequent is the curious sense that he is doing something wrong. There may be, in other cases, a more complete but more partial loss; there may be sudden darkness, for a moment, or a sudden silence, sometimes followed by sudden darkness, from the inhibition of the centres for sight or hearing, or both.

In a large number of cases the onset of the minor attack is accompanied by some sensation which the patient remembers. This must be ascribed, of course, to a discharge in the part of the brain with related function. There may be a flash of light, stars, a gleam of colour, or some more complex sensation such as has been described. This may precede loss or impairment of consciousness; the sufferer may even go on with his conversation, and others may be unaware of the attack. If there are also severe attacks the sensation may be the same as that which precedes these; but occasionally it is not the same, even if the severe fits are preceded by a well-marked aura. A common warning of both forms is the epigastric sensation.

Vertigo is a frequent momentary symptom of the minor attacks. It involves, by its nature, impairment of consciousness in so much as it depends upon an erroneous sense of the relation of the individual to his environment. It may be various in character, subjective or objective, but is of great importance on account of its frequency, and in that it is seldom associated with severer attacks in the same person. Indeed this is true of all varieties of minor epilepsy, and makes it necessary to put leading questions to ascertain their occurrence. The important characteristic is that they occur without any exciting cause; vertigo, for instance, may arise without any sudden movement to produce it. In all attacks in which there is loss of consciousness there may be micturition during the attack.

Local discharges, which cause the local commencement of convulsions that become general in severe fits, constitute, when they occur alone, the minor form of these attacks. If severe fits begin by clonic spasm in the hand, extending through the whole side and then to the other, the slight attacks consist of convulsion limited to the arm. Again, if the attacks begin with a sensation of tingling which passes up the arm and is followed by spasm, the minor form consists of the sensation alone, which often has the effect of inhibiting the related motor centre, so that the arm hangs powerless by the side. So likewise with the initiatory phenomena in other parts. When the sensation or slight spasm of such attacks occurs in the tongue or mouth there is often transient arrest of the speech centre.

Such slight local discharges, which do not spread beyond the centre in which they arise, are commonly unattended with loss of consciousness. Their frequency is often great, especially when they are due to an active morbid process which irritates, but progresses slowly. In such a case (of cerebral tumour) I have known 19,000 attacks to occur during 11 months.

Post-epileptic symptoms.—After a severe attack the patient lies exhausted and prostrated. For a few minutes the state of the spinal cord may be such that the knee-jerk cannot be obtained.[1] When it returns it is excessive, apparently from the lack of higher restraint; and after attacks of moderate severity there is commonly a foot clonus. It lasts longer on the side most paralysed, that is, the side towards which the head has been turned in the fit. A deep sleep follows, and if the patient be prematurely roused there is severe headache. In many cases there is little sleep but much headache. There is general sense of intense fatigue. Occasionally vomiting occurs before consciousness is recovered, or before the comatose state has passed into a sleep not too intense for the vomiting to awake the sufferer. Food which is then brought up may easily get into the larynx and cause fatal suffocation.

One other action after an epileptic fit, although rare, is important. A patient who is in bed or lying down, has occasionally a tendency, immediately after the fit, to turn over on the face. The post-epileptic slumber may be so deep that even the process of suffocation does not rouse him; asphyxia intensifies the commencing stupor, and death results. Rare as it is, this incident is of much practical importance. Whenever a person, supposed to be in good health, is found dead in bed, with his face against the pillow, death was the result of such suffocation after a fit. The inference is sure, even if the individual was not known to be the subject of epilepsy. The post-epileptic coma may also be intensified to a fatal degree by the administration of even $\frac{1}{4}$th of a grain of morphia before the fit. For this reason extreme care is necessary in the administration of morphia to epileptics, and if a fit follow an injection the patient should be carefully watched.

Automatism.—After slighter attacks, and indeed in proportion to the slightness of the attack, provided there be loss of consciousness, a peculiar state of automatic action is apt to ensue. This is apparently due, as Dr. Hughlings Jackson has suggested, to the withdrawal of the control normally exerted by the highest centres. It is not certain, however, that the abnormal action of the lower psychical centres may not originate directly; for attacks occur in which, however carefully observed, such automatic action does not seem to be preceded by any indication of a fit.

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[1] When a convulsion is unilateral, the resulting feebleness may be very conspicuous and last for a few hours. It has received the name of post-epileptic hemiplegia. It is important, however, to note that the sensory discharge may inhibit the motor structures, and it is probable that this element sometimes enters into the condition which is generally regarded as solely due to exhaustion.

The simplest form is that in which a person goes on with some action in which he was engaged at the time, and with every manifestation of consciousness, yet after suddenly recovering himself, cannot remember anything of what has occurred. Thus a man drove a waggon across London, and then found himself six miles from the place where he was, as it seemed to him, a moment before. The "solution of continuity" in the self-consciousness is as sudden as the restoration. This may occur after the slightest form of minor attack; or some simpler action may follow, unconnected with that which has preceded the attack—saying something which has no relation to the subjects engaging the attention, or muttering some unintelligible words. There is often some fumbling action of the hands, or some apparently purposive action which may be highly inconvenient. Taking off the clothes is especially common, perhaps from some vague sense of indisposition and the propriety of going to bed. A music master had to give up his profession on account of the liability to this equivocal proceeding when he was giving music lessons to young ladies, to whom the momentary attack was invisible. It is in this state that acts of violence may occur, as in the case of a patient who attacked a physician, in the out-patient room of the Hospital at Queen Square, with a poker. One man had a sudden slight attack, which caused him to stand still while crossing a crowded street. A passer-by took his arm and assisted him to the pavement, but no sooner had they reached it than the helper received a violent blow from the man whom he had assisted. In this state crimes may be committed, and when the excitement is pronounced the state has received the name of "epileptic mania."

Post-epileptic hysteroid convulsion.—This condition of brief insanity, the sequel of an attack of minor epilepsy, is pathologically related to another sequel. In those who are of the age and sex in which that state of the brain exists which underlies the peculiar symptoms termed "hysteria," namely, a few young men and most women, a slight and even a moderate epileptic fit may be instantly followed by hysteroid convulsion, quasi-purposive energetic movements, throwing about of the arms, kicking of the legs, arching of the back, varied with wild talking or manifestations of horror or fear. Such a state may last for a few minutes or for an hour or more. The initial attack is often unnoticed. Minor epileptic attacks in childhood, to which this sequel is added at puberty, are often then thought to have become severe epileptic attacks. These cases are extremely common, and almost always misunderstood. From the character of the obtrusive symptoms they are regarded as purely hysterical; yet whenever a patient has such attacks, recurring for years, it is practically certain that the essential disease is epilepsy, and the fact is of great practical importance. The difficulty of the diagnosis is, moreover, increased by the fact that, under the training which the brain thus undergoes, such patients may occasionally have independent purely hysterical attacks.

These cases are the only representatives of the hystero-epilepsy of the French which we meet with in this country. In the purely hysterical convulsion there is nothing, even in the initial stage, which an observer accustomed to epileptic fits does not recognise at once as wholly different. But the term "hystero-epilepsy" should not be applied to those of the post-epileptic hysteria. There is no justification for the use of the word in this country.

Excitants of attacks.—In idiopathic epilepsy attacks usually occur without obvious exciting cause. Nevertheless sometimes gastric irritation, an excess in alcohol, or sudden mental emotion, may induce a seizure. It is important to know that, even in true epilepsy, annoyance or a sudden fright may cause a fit. There is a strong but unjustified tendency to regard fits excited by emotion as hysterical.

Arrest of attacks.—The commencing local convulsion, or sensory aura, which is due to local organic disease of the brain, may often be arrested by a strong sensory impression on the limb above the part.[1] A ligature is the easiest and apparently the most effective; perhaps partly because the sensory impression is produced on the entire circumference of the limb. This arrest may be obtained even in cases of cerebral tumour. Apparently, the excitation of the sensory cells causes an inhibition of the motor cells in the path of the discharge, just as a sensory aura in the arm, too slight to excite discharge in the motor cells, may arrest their action and render the arm helpless.

In idiopathic epilepsy the warning seldom endures long enough to permit any measures for arrest to be adopted. Vigorous movement occasionally stops a commencing attack—an effect which may be due to a diversion or dissipation of some of the nerve energy ready for liberation of the motor structures of the cortex. A strong sensory impression may be effective in such cases also, especially one which acts upon the fifth nerve. Smelling-salts, a mouthful of common salt, or even a splash of water upon the face, may be sufficient.

The only available drug, which acts with sufficient rapidity, is nitrite of amyl. It is intelligible that the sudden flushing of the brain with arterial blood, and the sudden influence of the mechanical distension of the arteries, should disturb and arrest the abnormal action. It is only available when the warning is prolonged, and it does not always succeed. I have known its failure to be attended with some inconvenience from the suspicious presence of an empty bottle labelled poison in the hand of a man who had fallen down in the street and seemed on the point of death.

Course of epilepsy.—State.—Severe attacks occur in the waking state alone twice as frequently as in the sleeping state alone. About 0·3 occur in both waking and sleeping, about 0·2 in sleeping only, and nearly 0·5 only when the patient is awake. Minor attacks are practically confined to the waking state. Attacks which have occurred only in one state seldom change to the other, although the first attack may occur in the state in which subsequent attacks do not occur. Attacks which occur only in sleep may so change as to occur also in waking hours; it is rare for attacks which have occurred only by day afterwards to occur in the night also. Infrequent attacks during the night, in those who sleep alone, may go on for twenty years without their occurrence being suspected.

Menstruation.—It is common for attacks in women to occur either before, during, or after the menstrual period, simply because the nervous system is then generally disturbed. It affords no evidence of a relation to uterine or ovarian disease, of which indeed definite evidence is scarcely ever to be obtained.

Pregnancy.—In most women who are subjects of epilepsy, the fits occur during pregnancy with the frequency unchanged. Occasionally they cease during pregnancy, and if they have commenced during pregnancy they may sometimes be confined to that state for two or three successive periods. Afterwards they usually occur at other times. They do not seem to have much tendency to cause abortion. Attacks may or may not occur after delivery; when they do they seldom have any unfavourable effect [see next article "Puerperal Eclampsia"].

General course.—Puberty.—In very intimate connection with the occurrence of fits at the menstrual period is the relation to the course of the disease of the commencement of menstruation. When a girl has become subject to fits in childhood, the hope that they will stop on the establishment of the catamenia is generally entertained, and is sometimes encouraged by doctors. But we have seen that this period is one at which there is the greatest tendency for epilepsy itself to commence. The conditions favourable to its onset are not likely to be favourable to its cessation. This is entirely borne out by facts. If the disease does not cease before puberty it is not likely to be arrested for some years afterwards. In boys this period is far less definite, and it may be, in itself, practically neglected.

Tendency.—The facts of epilepsy show it to be a self-perpetuating disease, maintained and increased by a sort of habit developed in the "intimate function" of the brain. We have seen also that from what we know or conjecture of the minute processes which underlie these and other functional actions of the nerve-centres, it is easy to conceive that this self-perpetuation is inevitable. Thus the natural tendency is for attacks to become more frequent or more severe. And yet discerning, as we do, how much there is in the conditions of action and nutrition of the central nervous system that is quite unknown to us, we shall be prepared to meet with facts which seem inconsistent with our most careful inductions.

The disease often begins by minor attacks which may exist for years without severe seizures. The slight attacks may pass gradually into severe fits, or severe fits may commence and the minor attacks may cease. Conversely, minor attacks may replace convulsive seizures, and often do so as the result of treatment. In some cases, only too rare, all attacks may cease; we know not why. This event is scarcely ever spontaneous except in adult life. Since attacks go on through adult life, and the disease may begin at any period of adult life, it is clear that such cessation must be due to conditions and influences as yet unknown, which may neutralise the self-perpetuation, and may invalidate our forecasts.

The tendency to the cessation of attacks is greatest in the period between the completion of the structural development of the nervous system and the establishment of its chief functions; that is to say, in the period between the first three years of life and the second dentition. There is a slight tendency to cessation (manifested chiefly by the greater influence of treatment) between 10 and 13. In the following three years attacks are less likely to cease. After 16, however, treatment has a better chance of doing definite good. The more favourable conditions seem to persist through adult life, and at any time the malady may be arrested. There seems to be more prospect of arrest, however, between 30 and 45 than in later life.

It will be noticed that much of what is said of the course of the disease properly belongs to the prognosis. This is inevitable, because in the vast majority of cases the common treatment relieves when it does not cure. When it is intermitted the attacks become more severe or more frequent, or both. Observations on the course of the disease are thus of necessity observations on its course as influenced by treatment. Isolated facts come under notice occasionally which have a more definite significance, but they do not suggest inferences of much weight.

Frequency of attacks.—Severe attacks occur at intervals which vary from a few days to a year or even more. In the majority of cases the interval is between a week and a month. The interval is irregular in most cases. It is not uncommon for only one or two attacks to occur each year. But this fact is also true of the disease as influenced by treatment. In many patients the fits occur in groups; from two or three to ten or more may occur in a period of two or three days, and then no more for weeks or even months.

Minor attacks also present extreme variation in frequency. They may occur daily, and even many times a day; or at intervals of days, weeks, or months. In some patients they occur especially before or after a severe attack. They then become more frequent when severe fits are less frequent and evasive.

Status epilepticus.—The subjects of epilepsy, chiefly those who have passed early childhood and are not far beyond the completion of full development, occasionally have a series of fits in close succession. Before recovery of consciousness after one fit, another occurs, and the rapidly recurring attacks may go on for several days. The temperature rises two, three, or four degrees, and the patient is reduced to a state of extreme exhaustion, which may result in death. Fortunately this "epileptic state" is very rare. Nothing is known of its immediate causes.

Mental state.—The relation of mental disturbance to epilepsy is discussed in another article (vol. viii.), and the post-epileptic state has already been spoken of. Many sufferers from the disease are naturally weak-minded. The predisposition which underlies the instability of the motor and other structures, and is the remote and chief cause of the disease, is also manifested in imperfect function. It is not difficult to understand that this should be. But persistent mental feebleness may be a result of the attacks. Severe fits seldom cause it, but the result is common from minor seizures. It is not a necessary effect; several minor attacks may occur every day for years without the slightest influence on the mental power. We cannot yet say why the effect sometimes results. It especially follows attacks in which there is momentary loss of consciousness only, and this may be referred to the influence of the discharges on the structures of the brain which subserve the highest cerebral function; yet precisely the same attacks may occur for years with no apparent influence. The most important practical fact connected with mental feebleness is the frequency with which it is manifested when attacks cease, which have been going on for a long time. It is met with when the cessation is spontaneous, and therefore, although it is commonly ascribed to any medicine which has caused the cessation, its chief cause is probably the repression of the discharges to which the brain has been accustomed. Nerve energy must be evolved, ready for escape, as the result of previous liberation. If this escape be prevented, and yet the processes for the production of nerve energy continue, it is easy to understand that there may be a widespread interference with the activity of the brain. As far as we can perceive, medicines act by repressing the discharge, and it is only after a time that the energy for the discharge ceases to be generated. The depression of function of the brain influences the whole nervous system and the whole body. It is a temporary effect, it is often a source of undue concern to the friends, but it may be so severe as to necessitate an intermission of the treatment. After a severe convulsion the patient is at once better, as if a depressing cloud had passed away.

Causes of death.—Alarming as is the aspect of a patient in a severe attack of epilepsy, death very seldom results from the violence of a fit, and the danger in any given fit is negligible. This is true even of patients with grave heart disease. In the status epilepticus death does sometimes occur from exhaustion. When epilepsy ends life it is generally by indirect means. A fit may cause a fatal fall; it may cause death by suffocation in consequence of vomited food getting into the larynx, or through the patient turning over with the mouth against a pillow. Yet when all these causes are taken into account, it is remarkable how few persons die in consequence of this common disease. We have, indeed, imperfect knowledge of the causes of death of epileptics, but it is certain that even severe epilepsy may persist through life and may not prevent the attainment of old age.

Secondary forms.—We have seen that convulsions, however produced, become perpetuated by the influence on the cerebral nutrition and functional tendency of the recurring extensive discharges. By reason of this, the convulsions which are not idiopathic may become recurrent, after their cause has ceased, as a malady, indistinguishable from idiopathic epilepsy.

Reflex epilepsy.—One class of such secondary attacks is that which results from some peripheral irritation, and is called "reflex epilepsy." Convulsions thus produced are almost confined to the first half of life and to irritation of the gastro-intestinal canal. The most common cause is tapeworm. If they persist after the expulsion of the worm, they do so in consequence of the tendency established in the brain which cannot then be considered as essentially different from that of idiopathic epilepsy. Recurring convulsions from irritation of one of the cerebro-spinal nerves are extremely rare; and it is enough to say that the same statement is true also of these. Of epilepsy due to disease of the uterus or ovaries nothing is seen except by gynæcologists.

Toxæmic epilepsy.—The blood states which cause convulsions generally also cause death. It is possible that some chronic blood states, however, give rise to fits quite similar in character and course to those of idiopathic epilepsy. There is reason to think that such fits occur in some cases of chronic renal disease, and are due to a variety of toxæmia which differs from common uræmia. Lead poisoning may have the same effect. But of these events, also, it is often true that, if the patient recover from the blood state, the fits continue by the same induced bias of the brain as in the idiopathic disease.

Organic brain disease.—The convulsions due to active brain disease, generally to a tumour, cannot be regarded as properly within the subject of epilepsy. Nevertheless a very slowly-growing tumour may produce attacks which are difficult to distinguish from those of epilepsy, and are occasionally indeed indistinguishable from them. But stationary lesions of the brain are frequent causes of recurring fits. The disease is now and then a tuberculous tumour which has ceased to grow; occasionally such fits are due to old chronic meningitis on the cortex of the brain. In the vast majority of cases the lesion is a spot of old softening in the cortex, the result of a vascular lesion,—generally a thrombosis, which occurred in early life. If this involve the motor centres there is initial hemiplegia, partial or at first complete. But more often, as already stated, the lesion is not in but near a motor centre, and damages the gray matter only in slight degree. It is the slight damage, one which impairs power only slightly or not at all, which is the chief cause of subsequent discharges. Hence it is not often that such convulsions are met with in association with persistent severe hemiplegia; they may accompany slight hemiplegia, but most commonly there is only a history of initial weakness, or not even that.

The convulsions of this class are generally unilateral when slight, bilateral when severe. The secondary tendency to extension of the discharge through the brain, after a few years, may be so great as to cause the fits to become so quickly general that local or one-sided commencement is not observed. The effect of such repeated discharges is to induce a general state of the brain similar to that of idiopathic epilepsy, which may even assume the forms of minor epilepsy, such as momentary loss of consciousness, as in cases apart from any local lesion. This secondary state is further illustrated in an important manner by the fact that, after the convulsions have thus become quickly general, or minor attacks have been established, the removal of the part of the cortex from which the discharges proceed does not arrest the occurrence of the fits, or only does so for a time. Hence the removal does not justify any promise that the attacks will cease.

Pathology.—The chief inferences regarding the pathology of epilepsy have been mentioned in the introductory section. The nature of the disease can only be inferred indirectly from evidence, otherwise obtained, regarding the functions of the nervous system and from the negative effects of the disease itself. At present there is no *morbid anatomy* of the disease, nor is it likely that any conspicuous changes will be found, except such as may result from the excessive functional activity of the status epilepticus. In any case the effects of the attacks must be too widely distributed to afford any clue to the primary disturbance.

It would serve no useful purpose to describe the conceptions of epilepsy which have been current during the last forty or fifty years. They are no less interesting, and not much more instructive than the old demoniacal pathology which gave the disease its name. Thirty years ago it was generally believed that the epileptic fit was the result of a spasm of the arteries of the brain, due to the discharge of a vaso-motor centre in the medulla. Few minor conversions in the history of medicine are more remarkable than the absolute disappearance of this notion before the simple but conclusive facts which connected the manifestations of the disease with a morbid state of the cortex. They did this with such precision as to leave no room for any other mechanism. All previous hypotheses went down before the irresistible force of the facts which Dr. Hughlings Jackson saw first, and with the clearest vision.

(i.) Sudden liberation of nervous energy in the brain is one certain fact of the epileptic attack. (ii.) The first indication of this liberation which consciousness perceives proceeds from the cortex of the cerebral hemisphere. (iii.) We have no evidence that the initial symptoms ever proceed from any other part. (iv.) It is probable, then, that the nerve discharge which causes muscular spasm proceeds from the cortex of the brain. (v.) We have no evidence that the sudden liberation of energy in the cortex is the result of any preceding morbid action. (vi.) All that has been learnt of the action of the nerve-centres is in harmony with the primary disturbance being in the seat to which the symptoms point. (vii.) That which we know of the character of the action of the various nerve-centres is in harmony with the other indications that point to the gray matter of the cortex of the brain as the part deranged, in which part the nutritional effect of morbid function is the essential element in the disease.

Once more, if the explosion of nerve energy, as seems probable, arises in the cortex, it seems also to be probable that it does not arise in the nerve-cells. The recent advance in our perception shows that we must regard the spongy gray matter, in which nerve material is finely divided, and is adjacent to the ends of the branching processes of the nerve-cells, as that in which latent chemical energy is transformed into the nerve force which descends the nerves and stimulates the muscles.

Through the nerve-cells the impulses pass along the separate fibrillæ which, as continuous paths, traverse the cell and compose the axis cylinder of the nerve-fibre. To go farther into the region of speculative pathology would serve no useful purpose.

Diagnosis.—The diagnosis of epilepsy involves first the recognition of attacks, and, secondly, the recognition of their nature.

Occurrence of attacks.—(*a*) During sleep. A severe epileptic fit is a conspicuous event if it is observed. But, a solitary sleeper may know nothing of the occurrence of an attack, and it is important to remember the indications that one has taken place during sleep. They are—waking in the morning with a bad headache and a general sense of fatigue or soreness; evidence that there has been micturition in the night without the patient's knowledge, provided he is not a young child; considerable and unusual disarrangement of clothes; soreness on the side of the tongue; moisture or spots of blood on the pillow. The cases in which such evidence of night attacks are most likely to be found are those in which minor attacks occur in the day, the nature of which is clear, but generally unsuspected by the patient.

Minor attacks alone are likely to escape recognition. Often they are so slight as to be noticed only by the patient; if observed by others they are thought to be simply giddiness or faints. It is generally necessary to mention to the patient several common symptoms of such attacks, and to ascertain if he can remember experiencing any one of them. If sudden suspicious symptoms occur without reason, their significance depends largely on their occurrence when a patient is tranquil and not subjected to any disturbing influence.

To this, however, must be added suddenness of onset, brevity of duration, and that the return of the normal mental state seems to be through a brief fog. A sense of dazedness as the momentary attack is passing off is a very characteristic and important symptom. Other points will be mentioned in connection with special differences. It should never be forgotten that it is very rare for a patient or the friends to suspect that there is any relation between the minor and major attacks, or that the minor attacks have the slightest importance.

From hysteroid fits.—The convulsions of hysteria and epilepsy can scarcely be mistaken by any one who is acquainted with their features, and has an opportunity of observation. In hysteroid convulsions the spasmodic movements are such as might be produced by the will, they present what may be called a "purposive" aspect, quite different from the strong warping tonic spasm which constitutes the first part of a severe epileptic fit, and changes gradually to the clonic spasm of shock-like character. Nor is there in hysteroid fits the strong deviation of the head and eyes to one side, or the frothing of saliva, often blood-stained, from the lips; except possibly in rare cases of involuntary imitation of attacks that have been seen. If a severe epileptic attack begin with clonic spasm, this is local at the beginning, and the quick jerks spreading from the place from which they set out bear no resemblance to the regular less jerky bilateral muscular contractions of a hysteroid fit. In the former the patient has only to be kept from accidental injury; in the hysteroid attack forcible restraint is often necessary. The patient has to be "held down," and in this the essential difference in the character of the convulsion is revealed. Moreover, the duration of a single epileptic fit seldom exceeds three or four minutes, while most hysteroid attacks last a quarter of an hour, and often continue much longer.

A special diagnostic difficulty is involved in the occurrence of hysteroid attacks of characteristic form, as the immediate sequel to minor attacks of epilepsy. These may be so slight in degree as to be unnoticed, while the hysteroid attacks are obtrusive and are alone described. It is, however, common for such patients to have, at other times, attacks of petit mal without the hysteroid sequel. This can be ascertained by inquiry, although their relation to the other attacks is seldom suspected by the friends of the patient. It is also common for more severe epileptic fits, with tongue-biting, or micturition, to have occurred at some time, a history of which is conclusive. That which should always cause a suspicion of the real nature of the case is the course of the disease. Pure hysterical attacks do not recur in such a way as to constitute a prolonged disease. Whenever a patient has been subject to attacks, hysteroid in their obtrusive characters, for years, the malady is epilepsy with the hysteroid sequel to minor attacks.

It is most important to know the source of the fallacy which is due to the hysteroid condition superadded at puberty to the simple minor attacks of childhood. The account given by the friends seems to mean that previous attacks of petit mal have become severe convulsive fits. This sequence does occasionally occur, but in most cases of the kind there is only an addition of the hysteroid to the epileptic attack. The practical importance of the diagnosis is great. To do good the epileptic element must be treated; the hysteroid may be disregarded.

There is too little readiness to see that the tendencies on which "hysteria" depends may be called into activity by any disorder of the nervous system, by functional disease as well as by that which is organic, and by any kind of that disturbance which we call functional.

Vertigo is a common subjective symptom of a commencing epileptic seizure when loss of consciousness is not the first effect. When the vertigo is obtrusive it may be a question whether the attack be one of epilepsy or of auditory vertigo. The difficulty is increased by the fact that in rare cases this epileptic aura may be attended with a sudden noise in the ear. In auditory vertigo loss of consciousness is very unusual, and even impairment only occurs in attacks of extreme severity. In these there is vomiting and persistence of the vertigo long after consciousness has become normal, a symptom which alone excludes epilepsy. The characteristic of the giddiness which is associated with epilepsy is its occurrence without exciting causes; it occurs when the patient is still; the absence of excitation by movement is occasionally met with in aural vertigo, but its presence is strong evidence of the latter.

Syncope, simple fainting, is generally due to a definite cause, such as emotion, a hot room, and the like. It occurs in those who are deficient in strength. Loss of consciousness is not instantaneous, and on its return consciousness is perfect in character from the first, although its degree may only slowly reach the normal. There is not the "dazed" condition, which is a characteristic of the recovery from the minor epileptic seizure. Involuntary micturition never occurs during the unconsciousness of syncope, and is not rare in that of epilepsy. There is never any automatic action after simple syncope.

Other diagnostic problems occur so rarely that it is needless to discuss them, especially since they are for the most part included in this aphorism, that the great characteristic of epileptic attacks is suddenness of onset and brevity of duration. To this may be added spontaneity of onset, provided it be remembered that a sudden emotion may excite an epileptic attack in a patient whose seizures are generally without exciting influence.

Prognosis.—The prognosis of epilepsy is a question of therapeutics. That which we can perceive of its course and infer regarding its nature alike show it to be a self-perpetuating disease, in which no expectation of spontaneous cessation can be entertained. This conclusion is undisturbed by the fact that the disease may now and then cease spontaneously; this issue is so rare, and the conditions under which it occurs are so little known, that it cannot enter into a practical forecast.

The recovery from the disease is freedom from fits without treatment, yet in no disease does treatment enter into the prognosis to the same extent, or in the same degree. Hence in most of the cases in which the prognostic problem involves that of cure, the prognosis as to the malady is determined by the conditions of the patient and the possibility of treatment. In this disease, as in many other diseases of the nervous system, but more than in most, observation of the present alone can afford an accurate forecast of that which is to be. When every kind of attack is prevented, a patient is only on the road to a cure. As long as the slightest seizures occur, severe attacks would certainly occur were the patient to leave off taking the medicine which has arrested them.

But treatment may produce a state which, however far short of a cure it may be, often involves a vast difference to the subject of the disease. The ability to work, the capacity to enjoy life may be regained, and as a consequence the relief of the friends from the pressing anxiety that is involved in the frequent occurrence of a fit. By treatment epilepsy is sometimes cured, but life is often transformed; and how precious this imperfect result frequently is can only be discerned by those who have witnessed the gratitude it evokes.

The shorter the duration of the disease, and the less frequent the attacks, the brighter is the prospect of their permanent arrest. It must be remembered, however, that the less frequent the attacks the longer has treatment to be maintained before its influence can be seen, and therefore the more difficult is it to secure perseverance. The prognosis is better if attacks occur only in the night, than if they occur only in the day, or in both sleeping and waking states. It is better in idiopathic epilepsy than in the form which is the result of some old cerebral lesion.

The most unfavourable feature is the occurrence of minor attacks. These are often not influenced in the least by the treatment which stops the severe ones, indeed they may become more frequent. So long as they continue, if medicine is stopped severe attacks will soon recur. Hence the extreme importance of their recognition by the indications mentioned in the preceding section.

Treatment.—The tendency to the recurrence of convulsions, which constitutes the disease we call "epilepsy," can only be influenced by the administration of drugs; that is, by bringing certain chemical substances into artificial relation with the nerve elements concerned in the disease. The sketch of the nature of the disease which was given in the introduction should make the fact intelligible. Action is due to chemical union; instability to too ready union; stability is due to some unknown mode of restraint of the tendency on the part of the atoms of the nerve elements to unite with the oxygen brought to them in the plasma with which the nerve structures are bathed. From this comes the molecules which they appropriate for the maintenance of their nutrition and of their capacity for action. The nutrition of the elements and the replacement of lost elements is under the influence of life, and so also is the liberation of the latent chemical energy when atoms escape. The vital influence is beyond our scrutiny, the chemical influence we can dimly discern.

Mysterious as is the process, its control is not less so. The influence of the alkaline bromides, of potassium, sodium, and ammonium, is the most remarkable and definite therapeutical discovery of the last half-century, and it was purely empirical. It is within the experience of every student, that if a person who is having a severe epileptic fit every week begins taking a drachm of a bromide every day, he may not have a single fit for a year to come. Yet, during that time, there may have been no apparent perversion whatever of the normal action of the brain.

We have no evidence that the bromide salt is decomposed in the system in appreciable degree, or that its influence depends on any separate influence of the bromine. Indeed the proportion which its effect bears to the amount given is scarcely consistent with such an assumption.

We can discern no room in the constitution of the nerve substances for the assimilation by it either of bromine or of its salt. We may conjecture that the salt exerts its peculiar influence as such; as such, it restrains the ill-timed action, the wasteful liberation of nerve energy by dissolutions of the molecules in which energy lies latent. How this restraint is exerted we cannot comprehend. It may, however, be noted that such an influence of a salt is somewhat less difficult to understand if the finely-divided gray substance at the extremity of the dendrons be the seat of the transformation of latent chemical energy into nervous energy, than if we conceive the nervous energy to emanate from the nerve-cells.

The three alkaline bromides present little difference in the influence they exert. On careful comparative observation, we find it rare for a patient to do better on one than another; when such difference is met with, the greater influence seems to be in proportion to the amount of bromine in the salt: that salt which is most commonly found more effective is bromide of sodium; and bromide of ammonium sometimes seems to have a greater influence than bromide of potassium. It is, however, almost impossible to control therapeutic experiments of this kind. The difference is, moreover, of little practical importance. A combination of the salts has been recommended to prevent deleterious influence of the bases, but there is no evidence of such influence, and we do not know that there is the decomposition necessary to permit the base to have a special effect. It is strange, indeed, to note how little evidence there is for opinions that are widely acted upon. Bromide of strontium has been recommended and widely used, but I have been unable to perceive any evidence of the superior value of this salt.

Whichever bromide be employed, it must be given continuously; that is, the influence must be maintained without interruption. It is necessary to continue the regular administration of it for two years after the occurrence of the last attack of any kind, and then to spend another year in gradually diminishing the dose. Not until the end of the third year is it reasonably safe to omit the medicine altogether. This precaution applies to every method of treatment; there is no short method of cure. But the temptation to leave off the medicine is very great when the attacks have ceased; but it is then that the prospect of a cure makes persistence of the greatest importance. The duty of perseverance must be enforced upon the patient or his friends. Moreover, it is necessary to arrest all attacks, the slight as well as the severe: so long as minor attacks occur, it is practically certain that a severe attack will soon follow discontinuance of the treatment.

The bromide may be given once, twice, or three times a day according to the frequency of the attacks. A single dose will generally suffice if the interval between them is one of several months; but if it be less than two months two doses should be given. There is little difference in effect between two doses and three doses a day provided the amount of bromide administered is the same. Two doses of 30 grains have generally the same effect as three doses of 20 grains. A single dose may be given either at bed-time or after breakfast, according as the attack occurs in the night or in the day.

The object is to arrest the attacks, and also to afford a margin of stability sufficient to resist an occasional exciting cause. On the other hand, it is important that the dose given regularly should not be larger than is necessary to achieve this object. In many cases that which is necessary and not excessive can only be ascertained by experience in the individual case. But the largest daily quantity which, as a rule, can be continuously borne with impunity is 90 grains—half a drachm three times a day; and for many, perhaps for most patients, this quantity is large. The ordinary maximum may be put at 60 or 70 grains a day. Curable cases do not need more; and when more has to be given, the tendency to the attacks is so strong that bromide alone only relieves and cannot arrest them. Relief may indeed be almost as importan but it is unwise to strive after the unattainable.

The same daily quantity is sometimes better given in three doses. The difference in effect is slight, and is, on the whole, subordinate to the convenience of the individual patient. The system of increasing the dose when an attack is supposed to be impending is not to be commended. If such an increase be really necessary the constant dose is insufficient.

Some years ago I tried carefully the system of putting a patient through a course of larger doses of bromide in the hope of producing an effect on the nutrition and functional tendency of the nerve structures sufficiently great to be enduring. The dose and interval were increased until an ounce of bromide was given in nearly a pint of water every fifth day. A larger quantity was vomited. The result was not encouraging. After such a course of increasing doses, reaching the maximum at the end of three weeks, maintained there for about a fortnight, and then diminished through another three weeks, it was found that no lasting amelioration was produced. It was still necessary to use ordinary doses to prevent recurrence, and it was doubtful whether any more good had been achieved than would have resulted from the administration of ordinary doses for the same time.

Most persons can bear 60 grains of bromide daily for years without the slightest interference with the functions of the nervous system. The arrest of attacks is indeed often followed by some depression of the functions of the brain and of the general system, which depression may be ascribed to the bromide. But if this dose prove insufficient, and another attack occur, the depression immediately passes off. The action of the nerve-centres suggests that the repeated liberation of nerve energy in the epileptic fit induces the accumulation of latent energy, ready for similar release. This *preparation can only be prevented by long-continued* repression of its escape. But we also are compelled to believe that nerve energy cannot be prepared ready for instant release without an excess slowly passing off in what may be described as overflow (3). An abnormal amount, prevented from the escape for which it has been produced, must pass off in its overflow by other channels, and must thus derange the functions of those parts of the brain with which the centres are connected. Directly or indirectly this means the whole brain. It is important to realise this, in order not to misunderstand the effect which follows the arrest of attacks. The direct influence of bromide cannot, indeed, be excluded, but in most cases it is not the chief cause of the depression; the depression of function is in many cases an inevitable concomitant of the process of cure. But it is not enduring. It should be met by tonics; if these do not suffice, by a diminution in the dose of the bromide; only as a last resource shoul bromide be omitted, for this involves a risk of fresh attacks, whic are less easily arrested a second time. It may be necessary to encounter the disaster, but it can only be justified by the gravest mental and physical failure. It is important that this should be generally understood, because physicians are generally disposed to put all ill effects down to bromide, and to advise its discontinuance without any adequate reason; but the cessation of treatment may mean the destruction of the prospect of a cure.

But bromide has its drawbacks. It is not surprising that it should often interfere with the normal function of the brain; the marvel is that it should so often arrest the abnormal and leave the normal almost or completely undisturbed. We can conceive no difference between its action on the abnormal discharges which occur without recognisable stimulus and the normal discharges which are definitely excited. Yet a difference must exist, one sufficient to permit this agent to arrest the one and, in moderate but adequate doses, to leave the other free from appreciable interference. However, when doses of more than 75 grains a day are given continuously such interference becomes appreciable, and it is especially evident in the large number of cases in which the occurrence of epileptic fits is the expression of a general qualitative deficiency of the brain. In these cases the weakness of the intellect and backwardness of development manifest a state in which bromide cannot restrain the abnormal without interfering with the normal.

These are the patients in whom it is sometimes necessary to omit the medicine, and to permit the recurrence of attacks on account of the depression which is caused by the dose necessary to arrest the convulsions. There is no reason to believe that any other drug would be free from the same disadvantages: we have no reason to think that epilepsy could be cured by any agent except in the way that bromide cures it, and if we possessed any other means of influencing the essential process its drawbacks would be of the same kind.

Another drawback is the familiar influence of bromide on the skin. Foci of suppurative inflammation, in which the formation of pus is small in proportion to the inflammation, are the common effect; but the process may extend over a larger area, and give rise to a local dermatitis which produces a semi-purulent secretion, which dries into scabs like those of a rather thin ecthyma. In children large and deep ulcers may be formed on the limbs and elsewhere. Beyond the fact that bromine has been found in the contents of the pustules, we have no facts of significance regarding them. The tendency of the several bromides to produce this effect is in proportion to the amount of bromine they contain, but of the way in which the presence of bromine produces them we know nothing. Their occurrence, especially on the face and back, is doubtless due to the same conditions which make common acne most frequent in those situations. Patients prone to skin disease may have their own particular cutaneous affection evoked by bromide. On the other hand, some persons present a perfect immunity from these lesions. Although persons with thin skins may suffer from distressing irritation and congestion, the characteristic effects are chiefly seen in thick-skinned persons with abundant "comedones" upon the face. Why some persons never suffer is a problem which deserves more attention than it has yet received. The tendency to the acne rapidly diminishes after middle life; it is, on the whole, less in men than in women.

The only preventive of the bromide rash is arsenic, in the proportion of 5 minims of Fowler's solution to each scruple of bromide. No other expedient of any kind is even worth mentioning. In most persons the arsenic prevents the rash more or less, and in others it may reduce it to an endurable degree. But it must be continued as long as the bromide is continued, and it has its own evils. I have never known any general symptoms of arsenical poisoning to follow this treatment, but arsenical pigmentation of the skin is common, and sometimes we must allow the patient to choose between pigmentation and acne: never is there a moment's hesitation however intense the pigmentation may be.

Thirty-five years have passed since the influence of bromine on epilepsy became known, and for the latter two-thirds of that time its success and failure have alike stimulated an energetic search for that which should succeed when it fails, or to reinforce it when inadequate. The repertory of therapeutics in the pre-bromidic days, and all the pharmacological ingenuity of the present time, have been brought to bear upon the problem. The result is not unimportant, and yet it increases by contrast the mystery of that which is obtained by the simple administration of a simple salt.

But the influence of bromide is sometimes increased by the addition of other agents. Of these the most important are belladonna, in doses of 5 minims of the tincture, and digitalis in the same dose. At present it is not possible to give trustworthy indications for the use of either. It deserves note that in the West of England digitalis is an old popular remedy for epilepsy. The addition of either to an unchanged dose of bromide sometimes causes the attacks to cease. Indian hemp seems also to be occasionally useful.

Biborate of soda is perhaps the most effective of the drugs available in those inveterate cases in which bromide partially or wholly fails. It is one of a large number of drugs of which I have made careful comparative trials; it was first recommended by me in 1880. In doses of 4 to 10 grains three times a day, it often very much diminishes the frequency of attacks; but it does not seem to be capable of arresting them. In this incapacity it corresponds with all other drugs. The only drawbacks are trifling intestinal irritation, and a very typical psoriasis which readily ceases if arsenic is added.

Of the agents of repute in the pre-bromidic days, little need be said of the chief one, nitrate of silver. Thirty years ago patients with skins stained by silver were occasionally treated successfully by bromide.[1] We may reasonably conclude that silver does not offer us benefits commensurate with the inconvenience of it. The chief drug which has survived the introduction of the bromides is zinc. The lactate of zinc, advocated by Herpin, is often well borne in doses of 5 to 7 grains three times a day, and it seems sometimes to add definitely to the effect of bromide. It is inconvenient on account of its insolubility; this is true also of the citrate, which again I have tried extensively. The bromide of zinc is more soluble, but is not well tolerated by the stomach. On the whole the oxide causes least irritation.

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[1] Goulstonian Lectures. The fact is scarcely worth mention, but is referred to because its use seems to be thought, by most of those who have written upon it, to be a relic of antiquity.

The salts of zinc, however, seem to have more influence upon the minor attacks than on those that present convulsion. The influence is not great, but sometimes definite; and nothing is to be despised which can influence these seizures.

Belladonna and Indian hemp, also, in some cases have a definite influence on minor seizures; so likewise has morphia in doses of 5 to 10 minims of the solution. The hydrobromate of hyoscine is rarely useful. Nitro-glycerine again deserves trial in many cases, especially in children with frequent minor seizures; and in any cases in which symptoms of vascular disturbance are unusually prominent. The dose may be from ½ to 1½ drops of the 1 per cent solution. It may be combined with bromide provided some hydrobromic acid is added to prevent decomposition. Strychnine or nux vomica harmonises well as a tonic. The immense number of other therapeutic agents which have been extolled may be passed in silence. Most of them I have tried in vain, as well as a large number of drugs of which nothing has been heard.

Operative treatment.—It would be a waste of space to describe the various operations that have been advocated, whether on arteries or on the sympathetic nerves, "which have their day and cease to be": fashions which are not much to the credit of the profession.

One surgical procedure, however, stands on a different level. Trephining the skull is an ancient measure, undertaken ages ago, perhaps to let out an evil spirit. It has been revived of late to permit the exit of an evil substance. When the disease can be removed, and there is a reasonable prospect that the effects will not survive the primary cause, the operation is justified alike by reason and result. Such is the case when there is an irritating body which can be removed, be it a spiculum of bone or a stationary tumour. Thus it is in the case of a spot of softening, which causes only local discharges manifested by local convulsion. But when such disease, although removable, has so trained the whole brain into a habit of discharge that the attacks differ little from those of idiopathic epilepsy, the chances of the success of an operation are very small. A temporary arrest is followed by recurrence, and the only result is to add more or less hemiplegic weakness.

In cases of idiopathic epilepsy, trephining has been sometimes followed by cessation of the attacks, but the effect has been seldom proved to be permanent. Most cases are published before a sufficient time has elapsed to permit the effect of the operation to be estimated.

It is doubtful whether its action is other than as an energetic counter-irritation. Failures, often unregarded, are too numerous to bring the procedure within reasonable range, except perhaps in cases in which nothing is too grave, and no chance of good too small, to counterbalance the influence of the disease on the life of its subject.

In cases in which some disease in the region of the cerebro-spinal nerves is distinctly the starting-place of fits, and the disease is such as can be removed, it would be certainly right to rid the patient of the apparent cause, or to arrest its influence by resection of the nerve so implicated. Such cases may be met with, but they are extremely rare. The few cases on record have impressed themselves unduly on medical literature.

The status epilepticus seldom yields to bromide, or indeed to any drug except to chloroform, or to the hypodermic injection of morphia, or of hyoscine. The dose of morphia should be small, not more than $\frac{1}{12}$th of a grain, repeated, if necessary, every three hours. Hyoscine hydrobromate is sometimes effective, $\frac{1}{200}$th, $\frac{1}{150}$th, or even $\frac{1}{100}$th gr. being injected. Among other measures which deserve trial, however, are antipyrin and nitro-glycerine. The latter is less transient in its action than nitrite of amyl, and seems to change the functional state by flushing the nerve-centres with arterial blood. Inhalation of oxygen might also do good, but the means are seldom at hand.

Extreme care is necessary in the use of the hypodermic injection of morphia in epilepsy. I have already mentioned that death has occurred from the administration of a quarter of a grain shortly before the occurrence of a fit. The coincidence of the deep post-epileptic coma with the influence of the narcotic has been a sleep which knew no waking. Such cases are probably less rare than we suppose. The dose of morphia being within the common limits of safety, the cause of death may not be suspected. This caution is especially necessary in the status epilepticus, in which there is not the pain which seems, in so strange a way, to absorb the influence of morphia and lessen its narcotic effect.

General management.—The diet of the epileptic should be free from all that is obviously indigestible—raw fruit of firm texture, such as raw apples, the skin of dried fruit, including currants, pastry, except in small quantities, and tough meat. It has been recommended, on theoretical grounds, that meat should not be taken, but a careful comparison of a number of suitable cases for successive periods with and without meat, showed that the only difference to be observed was that the attacks were, on the average, a little more frequent and severe when meat was withheld. It may certainly be allowed with advantage, in moderate quantity, twice a day. But I have met with more than one patient who could not take beef without having an attack, although he could take other meat, and many patients find it wise to abstain from beef.

It is important that food should be taken slowly, and that it should not be too large in amount. Many young epileptics have a peculiarly ravenous appetite, especially before an attack, which should be restrained. Regular action of the bowels is of great importance, and should be secured by a daily mild laxative; it is unwise to take the risk of even one day's constipation.

The life of the epileptic should be as uniform as possible; irregularity in meals, overstrain in work, and excitement of every kind should be carefully avoided. Exercise is important, but definite exertion is harmful. And this rule affords also a guide to the mental training of the young, which is often an anxious matter. The education may go on, without injury and even with advantage, if all strain be excluded, and if it be remembered that the excitement of the playground may be more harmful than the mental work of the school. The same general rule applies to the life of older persons. The knowledge of the disease is sufficiently depressing to make it important that no other cause of depression should be added.

As little difference as possible should be made in the mode of life, but it is generally necessary to avoid crowded rooms and the vertiginous influence of the dance.

A knowledge of the nature of the malady is often carefully withheld from the sufferer. But such ignorance is undesirable if the needful care cannot be secured, unless the patient knows from what he is suffering.

Occupation.—The first point is that the occupation of an epileptic should be one in which the attacks shall not involve personal danger to life. This precludes a life at sea, on scaffolding, or about machinery. For the most part choice is restricted to sedentary occupations. The only wise rule is to assume that the attacks will persist, however good the prospect of their arrest. If this be done, the course of the disease is unimportant so far as the life-work is concerned; if it be disregarded, years of training may be wasted, and life may have to be recommenced too late.

Marriage.—The question of marriage of epileptics is one of great difficulty. The influence on the individual sufferer is quite unimportant. The chief consideration is the probability of the transmission of the disease, or the production of its congeners, insanity and idiocy. The subsequent influence of such transmission on its subject, on the happiness of the parents, and on the welfare of the race, have also to be thought of. When epilepsy is the result of an old organic cerebral lesion, however early in life this has occurred, it may be disregarded so far as transmission is concerned. The difficulty chiefly arises when there is evidence that the disease is distinctly inherited. Regarding the risk of transmission in such cases, the patient may be informed that in general terms there is no certainty that the offspring will suffer; that the chances are against the appearance of the disease in any individual child; but that they are also against the escape of one in every six children. With the statement of these facts the decision must be left to those immediately concerned. The general welfare of the community would preclude the marriage of any member of a family in which there is hereditary disease; but the welfare of the community has not yet a definite influence on personal action. Minute as must be the effect of any individual action, it is a part of that which makes for the well-being or ill-being of the race through years we cannot number. But "Utopia" has become a byword; the Present is urgent, and the Future seems remote. When uncertainty is added to distance, it is easy to evade that which is itself imperative.

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W. R. G.

PUERPERAL ECLAMPSIA

Definition.—This name denotes epileptiform convulsions coming on during pregnancy, labour, or childbed; and depending upon a disease of the kidney which is peculiar to pregnancy.

This definition excludes the fits of hysteria, epilepsy, gross disease of the brain, injury, or known poisons. Fits from one of these causes may occur during the childbearing process, but they have only an accidental connection with it. They are not eclampsia, and are to be distinguished from it (*vide* previous article, p. 781).

Frequency.—In pregnancy there is a special liability to kidney disease. The following figures will show this. The Registrar-General's report shows that the death-rate of women, aged from 20 to 45, from acute nephritis, albuminuria, and uræmia, is 4451 per million. The average number of children in a family, according to Duncan, is four. Assuming all women to be married, this would give three years, out of those from 20 to 45, passed in the pregnant state; and in this three years the deaths from the kidney diseases named would be 634 per million. As according to the last census 43 per cent of women between 20 and 45 are either single or widows, the incidence on the pregnant women would be about $\frac{4}{10}$ths of this figure, or 253 per million. Eclampsia, moreover, is very rare in the first half of pregnancy; and this would further reduce the estimated frequency of eclampsia, if pregnant women were not more liable to kidney disease than others. But the death-rate from eclampsia is 702 per million. Taking 20 per cent as the mortality, this would give 3510 per million as the frequency of the disease; which differs from most kidney diseases in that it often ends in recovery. Statistics of lying-in hospitals show that eclampsia occurs in about one labour out of 500. The Registrar-General's returns show that death from eclampsia takes place in one out of 2543 labours—a death-rate of about 20 per cent. Acute renal disease is therefore at least fourteen times more frequent in pregnant women than in women of corresponding age who are not pregnant.

The different renal diseases that may occur with pregnancy.—These diseases have been treated by many writers as one and the same; varying only in severity, and assuming in different patients different clinical aspects. But it appears to me that the cases fall into groups, and that the differences between these groups are broad enough to call for the description of each group as a distinct form of disease; that transition forms, intermediate between these different groups, are not found with the frequency that ought to occur if the disease were but one malady modified by conditions peculiar to individual cases; and, therefore, that the correct diagnosis and proper treatment of these different forms of disease will be promoted by keeping their differences clearly in view.

The diseases may be divided into four classes: (i.) The diseases that occur also in patients who are not pregnant. A patient with acute or chronic nephritis, granular kidney, lardaceous disease, pyelitis, or any other renal disease, may become pregnant; and the pregnancy may run its course without any great modification of the disease by the pregnancy, or of the pregnancy by the disease. I think such influence as the pregnancy has on these diseases is detrimental, but not so great as to be capable of demonstration. When renal disease of any kind coexists with pregnancy, the child is more likely to die in the womb than if the mother were healthy: but the proportion of children born dead from this cause is not great (29). (ii.) In pregnancy the pressure within the belly is raised: evidence of this is seen in the varicose veins and occasional œdema of the lower extremities. This raised pressure is not usually enough to produce an appreciable effect on the kidneys; but it may, when the uterus is very large, bring about the condition of the kidney that is seen with heart or lung disease obstructing the return of blood. This condition of kidney is well known; in pregnancy it seldom occurs, and it does not produce eclampsia. (iii.) A chronic disease peculiar to pregnancy. (iv.) An acute disease peculiar to pregnancy. The two latter I have now to describe.

THE CHRONIC RENAL DISEASE OF PREGNANCY.—**Definition.**—Saft has correctly defined the "pregnancy kidney," by which he means the chronic form of renal disease dependent on pregnancy, as follows:—"A disease of the kidney, which takes rise in pregnancy alone, never leads to important disturbance of the general health, and in childbed quickly subsides."

In some cases renal disease commencing during pregnancy continues to progress after delivery. I have published (33) a case of Bright's disease coming on during pregnancy, continuing after delivery, and ending in death nine months after the beginning of the symptoms—granular kidney and the cardio-vascular changes associated with it being found on necropsy. Such cases may seem to make the definition I have quoted a false one. But the cases in which kidney disease continues to progress after delivery are in my judgment instances of kidney disease different from that which depends upon pregnancy. They coincide with pregnancy, but do not depend upon it. For correct prognosis it is essential that the kidney disease which depends upon pregnancy should be distinguished from other forms.

Frequency of the chronic renal disease of pregnancy.—Different estimates of the frequency of albuminuria in pregnancy have been given, from that of Blot, who put it at 25 per cent, downwards. But the very high estimates have been made without distinguishing between pregnant women and women in labour; or between albuminuria with casts, and albumin from the bladder or the vagina, or the mere trace of albumin from the kidneys which is the result of raised pressure on the renal veins. Ingerslev took great care to eliminate these fallacies. He examined the urine of 600 pregnant women, and found albuminuria present in 29, or 4·8 per cent; but in only six of them (1 per cent) were casts present. Saft found albumin present in 5·9 per cent of gravidæ (never before the thirtieth week), and in 4·1 of multigravidæ. Casts were only present in a fifth of them.

Braxton Hicks examined the urine of 100 pregnant women (not stated whether in labour or not), and found albuminuria in 2; but one of these was a case of old kidney disease. Galabin (30) examined the urine of 13 pregnant women, but found albumin in one only. Hofmeier (38), from an examination of the statistics of the Berlin lying-in charity for 10½ years, out of 5000 cases, found 137 of nephritis, or 1 in 37; and of these 137 eclampsia occurred in 104; but it must be remembered that a lying-in hospital connected with a great school of medicine would be likely to receive an unusual number of bad cases. Hofmeier also examined 170 pregnant women for albuminuria, and found it present in two, or 1 in 85. Rosenstein estimates that about 1 in 4 of pregnant women with renal disease get eclampsia. Looking at these figures collectively I think we shall not go far wrong in roughly estimating the frequency of renal disease in pregnancy as about 1 per cent.

During labour albuminuria from pressure on renal veins is more frequent than during pregnancy. Ingerslev found that out of 153 women in labour the urine contained albumin in 50. Of these 50, 46 had also been examined during pregnancy, and albumin was then present only in 15. Saft found albuminuria present during labour in 32 per cent of primiparæ, and 22·6 per cent in multiparæ. Albumin is found in the urine oftener after the rupture of the membranes than before; and oftener when labour has lasted long than at the beginning of labour. In 80 per cent it disappears within forty-eight hours after delivery.

Clinical history.—The average age of the patients with this disease is above that of patients with eclampsia; and among them is not found the preponderance of first pregnancies that is observed with eclampsia.

The symptoms which bring the patient to the doctor are not striking: a little œdema, not of feet only; recurrence of vomiting, such as the patient had early in pregnancy; slight shortness of breath; some unaccustomed headache; weakness; more pallor than usual. These symptoms have come on so gradually, generally during the second half of pregnancy, that the patient can seldom give a definite date at which they commenced. The urine is generally lessened in quantity, but the amount may be about the average of health. If the quantity of urine is increased, the kidney disease is not that which depends upon pregnancy. It is clear, and not of high specific gravity; it may contain so much albumin that the urine becomes solid on boiling; but a more usual amount is from a quarter to half its bulk. The albumin is mainly paraglobulin; serum albumin suggests that the renal disease is not due to pregnancy. The urine deposits casts, usually granular. The amount of urea daily excreted is always below the average of health, even if the quantity of urine is not lessened. The degree of this diminution is important in prognosis: the more urea the patient excretes, the more likely is it that after delivery the renal disease will disappear. If, after due estimate of diet, the excretion of urea is greatly diminished, the albuminuria will probably persist after delivery.

Delivery is followed by diuresis. In patients suffering from the pregnancy kidney only, this diuresis is in proportion to the diminution of the amount of urine during pregnancy. The diuresis generally reaches its height from the third to the fifth day, and lasts about a week. At its height the amount of urine passed in the twenty-four hours may be twice or three times the average of health. With the diuresis there is an increase in the excretion of urea; the daily amount may be 600, 700, or 800 grains. The increase in the excretion of urea during the first week of childbed is greater in the patients whose albuminuria disappears than in those in whom renal disease persists.

Relation to eclampsia.—Although this form of kidney disease causes but slight symptoms, and ends usually in recovery, yet in probably about one case in five the acute disease which causes eclampsia becomes superadded to this chronic disease. For this reason the chronic renal disease is important.

Morbid anatomy.—We know nothing of the morbid anatomy of this chronic disease, for it is not fatal. The morbid changes in the kidney can only be found out by examining the kidneys of a pregnant woman who, having this disease, died from some accidental cause, and I know of no such record. A few necropsies have been published, but the accounts are not harmonious; for the evident reason that the patients who died were suffering from forms of renal disease such as occur independently of pregnancy, and not from that which depends upon pregnancy.

Treatment.—There is no drug that will cure the chronic renal disease of pregnancy; nor even one which will affect its course favourably.

The first thing in treatment is *rest*. This means that the patient is to be kept lying down in a warm bed, and protected from exertion and exposure by the attentions of a good nurse. In a patient so treated the albuminuria will almost always diminish, and the quantity of urine and urea will almost always increase.

French writers very strongly advocate *milk diet.* This means that the patient is kept on milk, and nothing but milk, from five to nine pints being given daily. The only other treatment is an occasional laxative to counteract the constipating effect of the milk. They relate cases in which improvement has followed this treatment, and in which eclampsia has been prevented by it. Tarnier says that he has seen the symptoms supposed to be prodroma of eclampsia disappear under milk diet, and return when the patient was put upon a mixed diet. I have seen albuminuria diminish while the patient was on milk diet, and increase again when beef-tea was allowed, the patient's circumstances being not otherwise altered. I therefore think that milk diet is beneficial and important in this disease, but I think it less important than rest. I have not seen accounts of cases treated by milk diet without rest in bed.

The *cure* for the chronic renal disease of pregnancy is the induction of premature labour; the main question, therefore, is whether this should be done or not. (As the symptoms are seldom well marked until the last two months of pregnancy, the question is usually one of premature labour, not of abortion.) If induction of labour be recommended, a delay of a day or two will usually be asked for, in order that suitable preparations may be made for the arrival of the baby; and therefore a trial of expectant treatment will generally have to be made before labour is induced. If during this trial of expectant treatment all the symptoms rapidly improve, the induction of labour may be postponed, or abandoned. But if, in spite of rest in bed and milk diet, the albuminuria and the accompanying symptoms persist, I think labour should be induced without delay.

The only objection to the induction of labour is the greater risk to the child's life from its premature birth. But with the help of an incubator a premature child may be reared without great difficulty. Were the pregnancy allowed to go on, the probability of the birth of a living child is not so great as it would be if the mother were healthy; for renal disease brings with it an increased liability to intra-uterine death of the child; and in eclampsia the fœtal mortality is high. But were the objection on the ground of the danger to fœtal life a stronger one than it is, I think that it should be disregarded in the face of the liability to eclampsia which threatens the mother if the chronic renal disease of pregnancy be allowed to go on.

A similar question may arise early in pregnancy, when a woman who already has some kind of kidney disease becomes pregnant. If the disease be slight, and the effect of pregnancy upon it not appreciable, there is no need to interfere. But if the kidney disease be enough distinctly to deteriorate the patient's health, and, above all, if it have got worse since pregnancy, I think that the induction of abortion, as soon as these points have been established, is good treatment.

A somewhat similar question is whether pregnancy should be allowed to go on in a woman who had one kidney removed. Twynam has collected cases from which to answer this question. The answer is that women after nephrectomy can bear children without very great danger, provided their organs and the remaining kidney are healthy. If the remaining kidney be diseased, I think that abortion should be induced.

THE ACUTE RENAL DISEASE OF PREGNANCY.—This is sometimes preceded for weeks or months by the symptoms of the chronic form of renal disease; and from them it may be inferred that the acute disease has attacked kidneys already the subject of the chronic renal disease. In other cases the disease attacks patients who have never complained of any symptom or shown any sign of disease, and whose kidneys are healthy. Dr. Handfield-Jones mentions a case in which normal urine was passed half an hour before delivery, yet within three hours' time puerperal convulsions manifested themselves, and bloody urine was passed, which on boiling became almost solid from coagulation of albumin.

In some such cases the patient has a fit without warning of any kind. In others premonitory symptoms of a kind different from those of the chronic form of renal disease precede the fit by a few minutes or hours. These prodroma are chiefly sensory. The patient may have *headache*; at first slight and occasional, then persistent and very severe, so that it may make her cry out with the pain. *Delirium*, restlessness, sleeplessness, or excessive somnolence and mental hebetude, may precede the fit. There may be *visual troubles;* asthenopia from failure of accommodation, amblyopia, diplopia, hemiopia, or amaurosis. Blindness may be sudden in its onset, and may be sudden, though it is oftener gradual, in its departure; in many such cases ophthalmoscopic examination has failed to discover any change in the retina to account for the amaurosis. Hecker attributed this transitory amaurosis to a retro-retinal œdema, interfering with the function of this membrane, which œdema may be absorbed, the normal state being then completely restored. There may be a sensation of light; thus J. Ramsbotham (56) mentions a case in which the fit was preceded by an exclamation that the room was studded with diamonds. *Acute epigastric pain*, sometimes so severe as to make the patient cry out, and accompanied with a sense of oppression and *shortness of breath*, are occasional premonitory symptoms. The latter symptoms may precede the pain. *Œdema* of the face and upper extremities, as well as of the feet, is often present; because the acute renal disease which causes eclampsia is often superadded to the chronic renal disease of pregnancy. But it is not a symptom characteristic of the disease which causes fits. Ingerslev, out of 71 cases of eclampsia, found no œdema in 15, and in 36 œdema of the lower extremities only; that is, in 51 cases, or 72 per cent, the kind of œdema characteristic of kidney disease was absent.

Acute renal disease without fits.—Sometimes, but rarely, patients have symptoms like those which precede and accompany eclampsia—headache, restlessness, giddiness, œdema, severe epigastric pain, amblyopia, and even stupor, together with the passage of a small quantity of urine solid with albumin—but without fits. There may be a few unilateral twitchings only, but no general convulsion; or there may be no convulsive movements at all. This condition may pass off during pregnancy, with or without death of the child, the acute symptoms lasting two or three days only, and the whole illness lasting about ten days (13, 34, 40).

Antecedent conditions.—(i.) The disease is especially frequent in *first pregnancies.* Thus Chantreuil (19) found that out of 683 cases, 522 were in first pregnancies, against 161 in multiparæ; and Schauta, out of 306 cases, found 253 in first pregnancies, against 53 in multiparæ. Schreiber found 109 first pregnancies, or 79·5 per cent, against 28 multiparæ, or 20·5 per cent.

(ii.) *Twin pregnancy.*—There is no doubt that eclampsia is more frequent with twin than with single pregnancies. According to Bartels, Litzmann, when doubtful whether a large uterus contained twins or not, was accustomed to decide against twins if the urine contained no albumin. Schauta out of 341 cases of eclampsia found 27 cases of twin pregnancies. Hofmeier (39) found out of 104 cases of eclampsia, 9 cases of twin pregnancy; Löhlein out of 103 eclampsia cases, 5 of twin pregnancy; and Schreiber out of 137 cases of eclampsia, 12 twin cases. Putting these figures together, they show a ratio of 1 twin pregnancy to 13 normal pregnancies, the proportion in pregnant women generally being as 1 to 80. According to Litzmann (as quoted by Bartels), this special liability to kidney disease does not occur when the uterus is unusually distended by dropsy of the amnion without twin pregnancy. Dropsy of the amnion, in single pregnancies, is so rare that it is difficult to test this statement. Nothing like eclampsia is ever met with in abdominal tumours—ovarian, uterine fibroids, etc.—which sometimes get much larger than any pregnant uterus. On the other hand, eclampsia has been observed with ectopic pregnancy. These facts indicate that the frequency of eclampsia with twin pregnancy arises from the presence of two children in the womb, and not from mechanical distension of the belly.

(iii.) *Reflex irritation.*—In patients liable to fits, any irritation or strain of the nervous system may cause the patient to have more fits than would otherwise have occurred. Hence in the etiology of eclampsia various causes of nervous disturbance have been assigned a place. Among them are fright, and emotional or physical shock; and depressing influences, such as poverty, anxiety, the disgrace of illegitimate pregnancy, digestive disorders, retention of urine, and exhaustion from prolonged labour. But eclampsia usually attacks patients who have not been exposed to any of these causes; and there is no evidence that they influence pregnancies in which eclampsia occurs any more frequently than those which end in normal labour. Their utmost effect is to make the patient have more fits than she would have had if in robust health, and it is doubtful if they do even so much as this.

It has been said that eclampsia is more frequent at certain seasons of the year, and also that it is sometimes epidemic; but these statements are not borne out by statistics, which show that, when large numbers are analysed, about the same number occur in each month (60).

Time of onset.—The disease may come on before, during, or after labour, but is most common during the first stage of it. Statistics show that the number of cases in which the fits come on during labour is greater than those, put together, in which they come on either before or after labour. Thus Charpentier (22) quotes a table from Wiegert of 455 cases collected from sixteen different authors. In 236 the fits came on during labour, in 109 before, and in 110 not till after. Schauta found the fits began during labour in 185 cases, in 42 they preceded it, and in 82 they followed delivery. Schreiber found 23 before labour, 85 in labour, and 29 after delivery. When the fits begin during pregnancy it is generally in the last two months. A few cases have been observed before this date, but they are rare. When the disease comes on during pregnancy, the attack is generally followed by the onset of labour, usually immediately, but sometimes not for some days; yet occasionally the convulsions cease, the patient recovers, and goes to term. When the disease occurs after delivery it is generally soon after it.

The fits.—These are epileptiform; that is, there is loss of consciousness, tonic spasm, followed by clonic, and then by coma. It is said that in eclampsia the cry which sometimes precedes an epileptic fit is absent; but, as by no means all epileptic fits begin with a cry, the distinction is not important. The spasms are generally bilateral, but commonly affect one side more than the other: I know no rule as to the side more often affected. Dr. Braxton Hicks observed the uterus participating in the muscular spasms. The duration of each fit is seldom longer than a minute. Cases have been described in which the fit is said to have lasted twenty minutes; but these are really instances of a succession of rapidly following fits. The interference of the fit with respiration is such that a fit which lasted longer than two or three minutes must be fatal by asphyxia.

The fit is never followed by an immediate return to healthy consciousness, but always by coma, which lasts a variable time—from a few minutes to several hours—and gradually passes into stupor, with restlessness and delirium, in which the patient often indicates that her head is painful. Sometimes increased restlessness precedes another fit. Sometimes the patient lies in persistent coma interrupted by fits. The coma is not a mere effect of the fit; for its depth and duration are not in proportion to the number of the fits. I have published a case in which the patient had two sets of fits: one while the urine was almost suppressed and was solid with albumin, and a second set when the renal function was partly re-established; in the second set of fits the coma was of short duration. The fits and the coma are alike effects of the disease.

In most cases there are not more fits than a single digit will express; there may be only two or three. On the other hand, it is said that as many as 160 have been counted (26). They do not recur at regular intervals. If the fits are many the disease is severe; in more than half of the cases in which there are over twenty-five fits the disease is fatal.

The urine (35).—(*a*) *Quantity.*—The urine is diminished in quantity to from one-twelfth to one-third of the average in health. The secretion begins gradually to be re-established in from six to twenty-four hours after the fits have ceased. In a few cases fits recur after the restoration of the urinary secretion has begun; but they are then not followed by such prolonged coma as when the disease is at its height. If the secretion of urine be not re-established, the patient dies. Observation of the amount of urine secreted is therefore important for prognosis. In a few cases the quantity of urine is not diminished, and may even be above the average of health; these are cases in which the acute renal disease of pregnancy has attacked kidneys already the subject of disease such as may occur independently of pregnancy. If such patients recover from the eclampsia, renal disease persists.

(*b*) *Specific gravity.*—Although the quantity of urine is diminished its specific gravity is very high—from 1030 to 1045. As the quantity of urine increases, the specific gravity decreases. In the exceptional cases, in which the quantity of urine is undiminished or above the average, the specific gravity is lower than normal—1010 or thereabouts.

(*c*) *Urea.*—The excretion of urea is largely diminished. In most cases not only is the quantity of urine diminished, but the percentage of urea in the scanty urine is lessened; yet in some the percentage is nearly normal, the urea excretion being diminished nearly in proportion to the diminution of the urine. The excretion of urea does not begin to be re-established until some hours after the cessation of the fits. Sometimes the percentage of urea in the urine begins to rise before there is any considerable increase in the quantity of urine; in others, the restoration of the urea elimination goes with increase in the amount of urine. In the exceptional cases, in which the quantity of urine is not lessened, the percentage of urea is so much reduced that the amount excreted is far below the average of health, even in cases in which the amount of urine exceeds the average. In some cases the elimination of urea is not re-established, but its amount goes on diminishing: in such cases the illness ends in death.

(*d*) *Albumin.*—In all cases the urine, at one stage of the illness, contains so much albumin that, when boiled, it becomes solid. In most cases the patient is not seen until she has had fits, and the urine is solid with albumin; in such cases we know not at what period this great albuminuria began. In Dr. Handfield-Jones' case the urine was free from albumin three and a half hours before the fits. In cases previously under treatment, whether for the chronic renal disease of pregnancy or for renal disease such as occurs apart from pregnancy, the urine does not become solid until the patient has had fits; and each fit is almost always followed by an increase in the amount of albumin.

It has been argued that in cases in which the fits come on without warning, in subjects apparently healthy, the albuminuria is altogether a result of the fits; but in disease, other than renal, which causes fits—in epilepsy, cerebral tumour, etc. — the fit or fits are either not followed by albuminuria, or the urine next passed contains only a trace of albumin. Such fits, even when they occur in pregnant women, are never followed by the passage of urine solid with albumin. But, as patients who are not aware of illness do not come for examination, I know no case of eclampsia in which the urine had been examined immediately before the onset of fits; it must be admitted, therefore, that there is no evidence which directly negatives this supposition.

The albuminous precipitate is composed of at least two kinds of albumin: serum-albumin and paraglobulin; and their relative proportion varies. The few observations that we have show that, in cases in which there is much serum-albumin and little paraglobulin the prognosis is worse than in those in which there is much paraglobulin. Serum-albumin is less diffusible than paraglobulin; hence it seems as if much damage to the renal cells is necessary to allow it to get into the urine in large quantity.

The distinction between the two kinds of albumin is made by saturating a specimen of urine with magnesium sulphate. This precipitates the paraglobulin, but not the serum-albumin. The specimen is freed from the paraglobulin by filtration, when the serum-albumin may be thrown down by heat and nitric acid. The precipitate so obtained is compared with the precipitate thrown down by heat and nitric acid from urine from which the paraglobulin has not been previously separated.

In cases which end in recovery, the albumin in the urine quickly diminishes after delivery, and usually disappears within a week, sometimes before. If eclampsia occurs in pregnancy, and, as sometimes happens, the patient recovers without labour coming on, then, after the fits have ceased to occur and coma has passed off, the albuminuria diminishes; but it does not disappear, as a rule, until the patient has been delivered.

(*e*) *Casts.*—In most cases the urine contains a few granular casts; in some none can be found; and in some the casts are many.

(*f*) *Sugar.*—According to Stumpf, there is acetone in the breath and sugar in the urine; and he explains the disease by supposing that the acetone irritates the liver and kidneys. I have in most cases found sugar to be absent; its occasional presence is not accompanied by any symptoms like those of diabetes, and is, I think, accounted for by the reabsorption of sugar of milk from the breasts: it is transient galactosuria, not diabetes (48).

(*g*) *Leucin and Tyrosin.*—These compounds are not spontaneously deposited from the urine in eclampsia; but Sir John Williams has published two cases in which they were sought for by chemical methods, and found.

Temperature.—During the height of the disease the temperature is usually raised, but not to a very high degree. A temperature over 103° is exceptional: its common range is between 100° and 102°. It fluctuates irregularly from hour to hour, variations which have not yet been reduced to rule. No association of other phenomena of the disease with particular variations in temperature has yet been shown. Winckel says that the temperature rises after a fit; but I have repeatedly found it to fall. In some cases, even though fits have ceased, the patient continues comatose, and the temperature goes on steadily rising until the patient dies (12). A temperature of 108° has been observed in such a case (31). Hypolitte has published charts illustrating this. Galabin has published a case of a series of epileptiform fits without albuminuria, proving fatal by hyperpyrexia, the temperature going up to 109°, in a child aged 11. It seems to me that such a mode of death is like the apoplectiform or epileptiform congestive attacks, attended, when fatal, with rapid rise of temperature, that are met with in various chronic cerebro-spinal diseases, such as sclerosis, general paralysis, old hemiplegia. But this is not the only mode of death. The temperature may fall before death, or it may be normal or subnormal throughout. Hence absence of fever is not sufficient ground for a favourable prognosis. I think, although I cannot adduce proof, that cases without pyrexia are those in which kidney disease was present before pregnancy.

Arterial blood-pressure.—Some have regarded this sign as of great importance; but we do not know whether in eclampsia it is a good or a bad thing for the blood-pressure to be high. Ballantyne has carefully studied it in three cases, so far as is possible, by the aid of the sphygmograph. His provisional conclusion is that in the first stage of labour with eclampsia, *even if the pulse be small the pressure is high.* During the fit the pressure is greatly increased, to fall again between the attacks. But if the first stage be prolonged and the fits frequent, the arterial pressure falls, and the pulse may become dicrotic, or even hyperdicrotic. There seems to be no close relation between the temperature and the blood-pressure. Ballantyne notes some variations in childbed which, if verified, are hard to understand; they may depend upon causes peculiar to the individual case: therefore I do not quote them.

Retinitis.—In some cases retinitis is present. In these cases chronic renal disease has usually been present before pregnancy began; and the disease persists after delivery. Miley has shown that with Bright's disease retinal changes occur late in its course, and affect prognosis; the mortality of cases with retinitis being double that of the cases without this complication. Eighteen months is an exceptionally long duration of life after retinitis has been recognised. From this it follows that retinitis with puerperal eclampsia indicates an unfavourable prognosis. But Bull of New York, while concurring with Miley in the unfavourable prognosis of renal disease with retinitis, says that in albuminuric retinitis, due to scarlatina or pregnancy, he has "repeatedly seen deposits completely absorbed and vision restored to normal." I have seen the albumin disappear after eclampsia with retinitis; but impairment of vision remained. Sir T. Grainger Stewart says "retinitis albuminurica is always a very serious symptom, except when it occurs in pregnant women."

Course and terminations.—Eclampsia seldom lasts longer than forty-eight hours. It generally ends either in recovery or death within that period.

(i.) Recovery.—About four-fifths of the cases end in recovery. The fits cease, and the patient gradually recovers from the coma which followed them. The secretion of urine begins to be re-established a few hours after the cessation of fits; but it takes from two to four days for the secretion to become as abundant as in health. After the patient has recovered consciousness, languor, mental dulness, loss of memory, and sometimes ocular troubles, persist for a time. Generally these effects pass off within a fortnight; but sometimes loss of memory lasts for two or three months afterwards.

(ii.) Death.—This may occur in one of several ways. (*a*) The coma may grow deeper and deeper, with or without cessation of fits, and the temperature steadily rise until the patient dies with hyperpyrexia. (*b*) The patient may die in continuous coma with a falling temperature, or with a temperature subnormal throughout. I think that the latter mode of death occurs in cases in which the kidneys were diseased before the pregnancy. (*c*) After eclampsia the patients always suffer for a few days from bronchitis, which is a result of the extreme venous congestion of the lungs during the fits. Sometimes more serious lung changes come on, and the patient dies, usually during the week following the eclamptic seizures, from capillary bronchitis or pneumonia. Schauta, out of ninety autopsies, found four cases of œdema of the lungs, nine of lobular pneumonia, one of hypostatic pneumonia.

The foregoing are the usual modes of death. There are rarer ones. (*d*) Cerebral or meningeal hæmorrhage has been found on autopsy oftener than I think can be accounted for by supposing it due to accidental complications, or to errors in diagnosis. Schauta found what was called "apoplexy" (large cerebral hæmorrhage being specified in four) recorded in ten out of ninety eclampsia cases examined after death. This frequency is explained by the greater liability of patients whose kidneys are already diseased—including those with granular kidney and degenerate cerebral vessels—to the acute renal disease of pregnancy. (*e*) Death may happen by asphyxia during a fit, in consequence of the fixation of the chest by tonic contraction of the respiratory muscles and the resulting arrest of respiration. (*f*) Bailly has published a case in which death took place by suffocation from swelling of the tongue, produced by hæmorrhage into its substance from the bites inflicted during the fits.

There may be more remote sequels. The fits themselves damage the brain. Puerperal insanity is more frequent after eclampsia than after normal delivery. Thus Charpentier, out of 203 cases of eclampsia, found 19 followed by insanity, or 9·2 per cent. The frequency of insanity in childbed generally, according to a table compiled by Kehrer, is about 0·2 per cent.

I have mentioned cerebral hæmorrhage as a cause of death. There may be bleeding into the brain, but not enough to kill the patient; if so, hemiplegia will persist. But this is a rare sequel.

Prognosis.—Death occurs oftener in multiparæ than in primiparæ. Schauta, out of 306 cases, found the death-rate among primiparæ to be 37·3 per cent; among multiparæ 44·9 per cent. The mortality is higher the earlier the fits come on. Thus, out of 309 cases, Schauta found the death-rate of those in which the fits preceded labour was 52·2 per cent; of those in which the fits came on during labour, 40·2 per cent; but of those in which the fits did not begin till after delivery, 27·2 per cent. These figures show a mortality considerably higher than that which usually occurs from eclampsia. I discover not from Schauta's paper the reason for this: his figures are based upon cases observed in hospital practice, and it may be that the table includes cases delivered in lying-in hospitals in pre-antiseptic times, or that cases of exceptional severity were selected for admission. Whatever the explanation, it does not affect conclusions based upon the comparison of different groups of cases all under similar conditions. There is no concomitant variation between the number of the fits and the death-rate; though upon the whole the death-rate rises with the number of the fits. Thus in most cases the fits do not exceed ten, and the death-rate is about 20 per cent. Of those in which the fits exceed twenty in number, more than half end in death. Of those in which the fits exceed fifty in number, almost all so end.

The risk to the child.—The fœtal mortality is high. The lowest estimate that I am acquainted with, taking all cases together, is 30 per cent. Charpentier (23) puts it at 44 per cent; Charles, basing his estimate on statistics derived from Belgian maternity hospitals, at 42 per cent; Tarnier at from 60 to 77 per cent. Schreiber found a fœtal death-rate of 26·1 per cent. Schauta has shown that the fœtal mortality, like that of the mother, rises with the number of the fits. He found the fœtal mortality, in cases in which the number of fits did not exceed ten, to be about 14·3 per cent; while of those who had more than twenty fits, more than half the children were born dead. This high fœtal death-rate is chiefly due to asphyxia during the fits; partly also to the frequency of premature delivery and the unusual number of twin pregnancies. The mortality that is due to the effect of the fits upon the child will obviously be reduced to a minimum by the extraction of the child as early as possible, while it is alive. I shall consider subsequently whether this be good practice or not; I only point out here that it reduces fœtal mortality; and the widely different results as to fœtal mortality that I have quoted very much depend upon whether this practice was followed or not.

Influence of the death of the fœtus.—It has been said that the death of the fœtus, when eclampsia is present, produces a change for the better. This was stated by Winckel (74), and has been emphasised by Byers (18), who says he has frequently verified it. But these writers have not detailed the facts upon which their clinical inference rests. Dr. Barbour has related a case in which rapid diminution of albuminuria and disappearance of œdema coincided with death of the fœtus in utero; and he quotes three other cases observed by Underhill, Maclaren, and Spiegelberg, showing the same thing. He rightly says that four cases are not enough to justify a conclusion; but if further observations should show that the concurrence of death of the child and diminution of albuminuria is more than a coincidence, a reasonable inference would be that the albuminuria is due to a condition of the maternal blood induced by pregnancy. On the other hand, Schreiber (62) has published four cases in which the disease came on in women who were delivered of macerated fœtuses, which must have been dead long before the eclampsia.

The effect of delivery upon eclampsia.—When fits begin before labour, they may either cease before labour comes on, or continue until delivery, or after it; and the disease follows the one course about as often as the other. But when fits begin during labour they continue in most cases after delivery. Schauta, out of 185 cases, found that fits ceased with delivery only in 62, or 33·5 per cent; in most the fits increased in frequency after delivery. Bruumerstadt, out of 105 cases, has reported 63 which show the same thing. Schreiber, out of 105 cases, found that fits ceased after delivery in 64·7 per cent, and persisted in 39·2—not a great difference.

The practical importance of these figures is that they show that delivery does not influence the course of eclampsia favourably; and that consequently operations to expedite delivery are not beneficial to the mother. The advice sometimes given, to "deliver as quickly as possible," the *accouchement forcé*, that is, the tearing or cutting open the genital passage instead of waiting for its natural dilatation, ought in my opinion to be rejected; for such measures increase the danger, both of death during delivery or childbed and of chronic invalidism afterwards. The mother's welfare should be preferred to that of the child; she should not be exposed to these risks simply that a living child may be born.

Morbid anatomy.—Our knowledge of the morbid anatomy of puerperal eclampsia is behind that of its clinical phenomena. This is because most cases of this disease are observed by persons who practise midwifery, and who are not, therefore, in the habit of making necropsies. Of the few descriptions of the kind many are imperfect.

I take first the organs most plainly at fault, namely—

The kidneys.—Very different conditions of kidney have been found. Bouffe de St. Blaise (11), speaking from an examination of 26 cases, says "there is nothing fixed in the lesions observed." Leyden says that the conditions of the kidney vary—"sometimes large, sometimes red, sometimes pale, sometimes granular." A comparison of the different accounts that have been published shows that the conditions of kidney present may be divided into three groups—

(*a*) Disease of the kidney, such as may occur apart from pregnancy, may be found after death from eclampsia. Any damage to the kidney makes it more liable to the acute renal disease of pregnancy. Thus Schauta, out of 90 necropsies in cases of eclampsia, found 2 cases of lardaceous disease, 2 of hydronephrosis, 4 of dilatation of the ureters and renal pelves. Lohlein, out of 32 cases, found once suppurative nephritis, once wasting of one kidney, and eight times dilatation of the ureters and renal pelves. It will be seen that dilatation of the ureters and renal pelves occurs often enough to justify the belief that it may have some influence in bringing about the disease, but not with the regularity necessary to make it an explanation acceptable for all cases.

(*b*) In most cases there are changes in the kidneys which to the naked eye resemble those of acute and chronic tubular nephritis. Schauta found this condition in 46 cases; 28 of these were said to be chronic nephritis, 16 acute, and 2 acute upon chronic nephritis. Löhlein, out of 32 cases, found parenchymatous nephritis in 17, and in 12 a mixed form of parenchymatous and interstitial nephritis. Wieger found the lesions of Bright's disease in 26 out of 27 cases. But although the naked-eye appearances are like those of nephritis, the few microscopical examinations of such kidneys that have been made lead me to think it doubtful whether nephritis was present.

(*c*) In some cases the kidneys have shown no sign of disease appreciable by the unaided senses. Out of Schauta's 90 autopsies, in 7 it was expressly stated that the kidneys were normal, and in 23 others the state of the kidney was not mentioned; in these latter, I think, it may fairly be assumed that the morbid changes in the kidneys were not very marked. A writer, quoted by Bouffe de St. Blaise,[1] out of 6 autopsies found the kidneys normal in 3. Out of 3 autopsies reported by Winckel, in 1 it is said that hyperæmia was the only morbid change shown by the kidneys.

Microscopical changes.—When these kidneys, either those looking like kidneys with acute or chronic nephritis, or those which look healthy, are examined with the microscope, the changes found are not those of inflammation, but a degeneration like that which is seen in blood poisoning. Angus Macdonald was one of the first to perceive the importance of this. The kidneys of one of his cases were examined microscopically by Professor Hamilton of Aberdeen. He says: "Judging from the naked-eye appearances, we expected that parenchymatous inflammation of the tubular epithelium in the cortex, passing into a state of fatty degeneration, would be revealed. The first glance at a section of the organ, however, showed conclusively that this was not the case, and that the lesion was not an ordinary parenchymatous inflammation." It consisted of "degeneration of the epithelial cells of a certain proportion of the tubules in the circumferential aspect of the cortex. The degeneration appeared to be of a colloid nature. The products of these degenerated cells ran down and blocked up, more or less completely, the other convoluted and straight tubules, so as to render them functionally useless, although their tissues were not diseased." In a case of my own (36), sections of the kidneys described by the late Dr. James Anderson, from naked-eye examination, as being the subject of "parenchymatous nephritis," were prepared and submitted to my colleague Dr. Charlewood Turner, who reported as follows:—"They show recent degenerative changes of the cortex, without any older or cirrhotic lesions—changes attributable to some toxic matter in the blood. Granular degeneration and swelling of the epithelium of the convoluted tubes, the nuclei of which are invisible, and swelling of the connective tissue throughout, without infiltration of leucocytes. In the medullary tissue the epithelium of the loops and connecting tubes appears normal. Some of the loops contain casts. There is much vascular congestion in this part. The glomeruli appear normal. There is no exudation in the capsules." Cutler has reported a case of eclampsia with necropsies in which the kidneys were examined microscopically by Dr. W. J. Fenton. His report is: "Most tubules show what is apparently a running together of the cells, having no definite structure, and having their nuclei obscured, the whole condition of the epithelium being similar to that often found in acute toxic conditions." Winckel (75) has reported a case in which the kidneys were examined with the microscope. They showed finely granular degeneration of the epithelium of the renal tubes, mostly in the cortex. Schmorl (61) found necrosis of renal epithelium. In a case of Leyden's the kidneys showed, "not nephritis, but the so-called second stage of fatty degeneration." Winkler (77) in nine autopsies found fatty degeneration of renal epithelium in all. Bouffe de St. Blaise sums up the histological results as follows:—"The characters closely approach the different coagulation necroses found in infectious diseases. The lesions fall principally on the epithelial cells and the vessels immediately in contact with them."

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[1] Indicated by a pronoun, the antecedent sentence not making it clear to whom the pronoun refers.

From these different observations, made by independent pathologists, having no hypothesis to support, it follows that, although the kidneys often look like inflamed kidneys, yet that the lesion of puerperal eclampsia is not nephritis, but an acute degeneration such as is caused by blood poisons.

The liver.—For reasons already given, there are but few autopsies of eclampsia cases on record in which the liver was carefully examined. But these few observations agree as to the nature of the changes present. The most careful study of the changes has been made by Pilliet and Létienne. The most conspicuous change is the presence of hæmorrhages into the substance of the liver. These are generally seen as dark red points and blotches. They may be so slight as not to be visible to the naked eye, but when none is thus seen the microscope reveals their presence. There may be great hæmorrhage, so great as to strip off the capsule, burst through it, and escape into the peritoneal cavity. The hæmorrhages are most abundant near the suspensory ligament. Between the red stains the liver is yellower than normal, and on microscopic examination the hepatic cells show either fatty degeneration or necrosis such as occurs in acute infectious diseases. The red stains caused by hæmorrhage and the yellow colour from degeneration give the liver on section a marbled appearance. The process seems to be one of multiplication of the nuclei followed by degeneration of the cells. The above description is based on sixteen autopsies, with microscopic examinations, made by Pilliet. It will be seen that they are something like the changes observed in acute atrophy of the liver. Schmorl (61) in fifteen autopsies, and Winkler (74) in nine, found in every case changes in the liver like those described by Pilliet. In a case published by Sir J. Williams (73) the liver was examined—"it was somewhat enlarged, and presenting vacuolated cells and disseminated points of necrotic tissue." He says: "The conditions presented by the liver can only be the result of severe contamination of the blood." Hypolitte has reported cases in which changes like those described by Pilliet and Létienne were found in the liver.

The spleen.—This organ is swollen and shows hæmorrhages under its capsule and into its substance. The hæmorrhages may be so great as to strip off the capsule extensively, or to reduce the splenic substance to a pulp. *These changes are like those which take place in the liver.*

The lungs.—These are always congested, with ecchymoses underneath the pleuræ. There are often pulmonary apoplexies, and sometimes blood in the bronchial tubes.

The heart.—The right side of the heart always contains clot. The hypertrophy of the heart (which it is now generally agreed takes place in pregnancy) is said by Löhlein, speaking from 16 autopsies in which the heart was weighed, to be greater in pregnancies with eclampsia than in pregnancies without eclampsia. As, apart from pregnancy, renal disease goes with hypertrophy of the heart, this is not surprising.

The brain.—In describing the clinical history of the disease I have referred to the occasional occurrence of cerebral hæmorrhage. But as a rule the central nervous system shows only such vague changes as "hyperæmia," "congestion," "anæmia," "œdema," "serous effusion," etc., the significance of which I shall point out in considering the theory of the disease.

Fat emboli.—Virchow, in four cases of eclampsia examined after death by him, found fat emboli in the lungs and in the glomeruli of the kidneys. He was unable to explain whence the fat comes; indeed, he does not think there is any relation between the fat emboli and the other phenomena of the disease. His discovery, therefore, does not instruct us much; but a fact in the disease vouched for by this great pathologist deserves mention. Winkler (77) found fat embolism of the lung in one case.

Pathology.—We know not the cause of eclampsia; therefore there has been much speculation about the nature of it. Hypotheses long current call for consideration: therefore I will not pass them by without giving my reasons for thinking them inadequate. For neurologists they may be extinct, but they often reappear in case reports, and even in text-books on midwifery.

The oldest views ascribed the disease to certain brain conditions:

anæmia, congestion, or œdema of the brain. If any one of these inconsistent morbid states were proved to be constant in eclampsia, it would not carry us far in the explanation of the disease; for we should have still to ask why the brain was anæmic, congested, or œdematous, as the case might be.

Anæmia of the brain, especially of the medulla oblongata, has been adopted as an explanation for the following reasons:—(i.) On post-mortem examination it has been thought that the brain looked anæmic. But whether the brain looks anæmic or congested depends upon whether the chest or the skull be first opened. If the chest be opened first, and the great veins cut, the blood drains out and the brain looks anæmic; if the skull be opened first, the cerebral vessels are full and the brain looks congested. Even if this error were guarded against, anæmia of the brain would only show its condition at the time of dying, not its state at the beginning of the disease. (ii.) That at the beginning of the fit the face is pale. But Moxon showed that one of the first phenomena of an epileptiform or epileptic seizure, in almost every case in which the point has been investigated, is a temporary stoppage of the pulse. This is a part of the fit, and it sufficiently accounts for the paleness of the face. If we assume that this cessation of the heart's action causes anæmia of the brain, and that the anæmia of the brain causes a fit, we still have to explain why the heart stops. If cerebral anæmia explains the phenomena of the fit, it explains not why the fit occurs. I think it does not explain the phenomena; for in ordinary syncope we have the same pallor and slowing of the heart, but an epileptiform fit is not the result. (iii.) That in animals ligature of the carotid and vertebral arteries produces fits, and contraction of those vessels, by spasm of their muscular fibres, has been supposed to act in like manner. But Moxon showed that vaso-motor nerves cannot contract vessels anything like as much as a ligature; and that the contraction effected by vaso-motor nerves is too slow and gradual to account for the suddenness of an epileptiform attack.

Congestion of the brain.—The fits have been attributed to "congestion of the spinal cord"; but as they affect the face this is obviously an insufficient explanation. "Congestion of the brain and spinal cord" is one of the oldest notions, dating from Mauricean. The lividity of the face and the fulness of the veins in the neck leave little doubt that during the fit the brain is congested; and the liability to loss of memory and insanity after eclampsia may be explained by damage to the brain by this congestion. But congestion in the fit proves not that congestion caused the fit. Moxon showed that in the morbid conditions attributed to congestion of the brain (epilepsy included) there is no evidence of such congestion; and that where congestion—that is, an excessive determination of blood to the brain—is undoubtedly present, there is neither epilepsy nor any disturbance of brain function whatever. [*Vide* art. "Circulation of the Brain," p. 239.)

Œdema of the brain.—This is known as the "Traube-Rosenstein" theory. According to this view the primary factor is a watery condition of the blood. I do not know that the blood of eclamptic patients has been proved to be more watery than that of other patients; but I pass by this objection. From the watery condition of the blood results a transudation of serum, which may be aided by the high blood-pressure usually present in eclampsia. The exuded serum makes the brain œdematous; and the swollen tissue squeezes the blood-vessels, thus producing anæmia of the brain, from which springs the eclampsia.

Besides what I have said in considering the relation of anæmia of the brain to fits, there are two grave objections which prevent me from accepting this supposition. The first is, that when the exudation reached such a degree that the œdematous brain tissue compressed the vessels, the pressure outside the vessels would be greater than the pressure within them; and as soon as this became the case transudation would stop. The second is, that transudation from the vessels would go on in other parts of the body as well as in the brain—indeed, to a much greater extent than in a cavity bounded by an unyielding bony case: and we should find the tendency to eclampsia to be in proportion to the amount of œdema. The reverse is the case; in many cases of eclampsia there is no œdema.

Exalted nervous tension.—Another notion, received with laudation at the time, we owe to the facile pen of Dr. Robert Barnes; it is that during pregnancy there is a state of "exalted nervous tension," a storing up of nerve force for the purpose of delivery, which in some subjects, owing to a hydræmic and poisoned condition of the blood, bursts out in a sort of explosion before delivery. We have no way of measuring "nervous tension" or nervous force, and we cannot say whether these things are exalted or increased, or the reverse; and "exalted nervous tension" is, therefore, a phrase empty of meaning, which only obscures the subject.

The toxic theory.—I cannot put this more clearly or more tersely than it has been put by Charpentier (25), "the urine of healthy people is highly toxic." On the contrary, the urine of certain sick people is not. As physiology authorises us not to consider the different poisons contained in urine (Bouchard has isolated seven) as products elaborated in the kidneys, these poisons must be in the blood. But toxic products cannot be present in the blood without the organism suffering. They ought to be eliminated in exact proportion to their formation in the blood; and this elimination is done by the kidneys in the urine. If the blood is not normally toxic and we are in health, it is because the urine is normally toxic, and incessantly takes from the blood its toxicity.

While the kidneys act regularly, the elimination of toxins from the blood is regular and incessant; and from this equilibrium results the normal condition—the state of health. If this equilibrium be disturbed, the organism suffers and disease is produced.

In the individual thus diseased two conditions may occur:—

1. There may be excessive production of toxic matter.
2. There may be insufficient elimination by the kidney.

In the albuminuric patient the urine is much less toxic than in the normal state, and in the eclamptic the toxicity of the urine hardly exists. In the latter the two conditions above mentioned concur with their maximum intensity. In them there is, on the one hand, diminution of the urinary secretion, which may go almost to anuria; and, on the other hand, absence of the toxicity of the urine with arrest of the elimination of toxines. From this arrest of elimination comes accumulation of the non-eliminated toxins in the blood, and appearance of the phenomena known as uræmic, which always coincide with the disappearance of the toxicity of the urine.

This accumulation of toxins in the blood of albuminurics is to-day a fact. Announced in 1886 to the Society of Biology by Doléris, it has been confirmed by the observations of Tarnier and Chambrelent, who have not only proved that the toxicity of the blood-serum in puerperal eclampsia is very considerable, but, further, that this toxicity of the blood serum is in inverse proportion to the toxicity of the urine.

We have the following evidence in support of the above statement:—

Doléris and Butte extracted from the blood of eclamptic patients a crystalline substance possessing toxic properties—capable, that is, of quickly killing rabits, guinea-pigs, and sparrows. But we know not yet the nature or the mode of production of this substance.

According to Bouchard (9), the average toxicity of the urine is about 45 cc. per kilogramme; that is to say, that a rabbit can be killed by injecting into it 4·5 per cent of its weight of the urine of a healthy man. This estimate is liable to variation one way or another. Bouchard puts 3 and 6 per cent as the limits of normal variation; that is, that 3 per cent of the animal's weight of some healthy urine will kill it; while 6 per cent of some other urine, also from healthy persons, may be required to cause death. The toxicity of the urine varies at different times of the day; the above figures apply to the whole urine passed during twenty-four hours. Laulanié and Chambrelent found that the toxicity of the urine of healthy pregnant women is much less than that of urine from healthy individuals not pregnant. They made six experiments, and found a toxicity represented by the figures 5·5, 6·9, 9·5, 10, 12·5, and 15,—all of them less than the average estimate of Bouchard for that of health. They hold that this diminished toxicity of the urine indicates that the organism of the women at the end of pregnancy must be more or less saturated with toxic matters. Blanc has performed thirteen similar experiments, and found that the average toxicity of the urine of women in the last two months of pregnancy was 7·6 per cent—a result agreeing with those above quoted in showing a less toxicity than that of health. He also made eight experiments with the urine of lying-in women, and found that in them the average toxicity was 5 per cent, or very nearly that of health.

Tarnier and Chambrelent examined the toxicity of the blood-serum in two cases of eclampsia. They found the toxicity of the blood-serum to be from 3·3 to 4·3 per cent; that of healthy blood-serum being about ten per cent.

Ludwig and Savor have experimented with (*a*) the urine of eight healthy women in labour; (*b*) the blood-serum of twelve healthy women in labour; (*c*) the urine of sixteen cases of eclampsia, (*d*) the blood-serum of sixteen cases of eclampsia. They show (i.) that the blood-serum of pregnant women is more poisonous than that of women not pregnant; (ii.) that the urine of pregnant women is less poisonous than that of women not pregnant; (iii.) that the blood-serum of eclamptic patients is more poisonous than that of healthy pregnant women; (iv.) that the urine of eclamptic patients at the time when the blood toxicity is at its highest, is much less poisonous than that of healthy women; and (v.) that in eclamptic patients who survive, there is a period after the fits have ceased during which the toxicity of the urine is much increased.

If the statements made in the above paragraphs be correct, whence come the toxins? and why are they not eliminated?

The formation of these toxins is a question of physiological chemistry which I am not competent to discuss. Bouchard holds that they are formed, (i.) in the tissues; (ii.) in the secreting glands; and (iii.) in the bowel—the latter poisons being of two classes. (*α*) some ingested with food; (*β*) some formed by decomposition of food within the bowel The latter opinion is important because upon it a scheme of treatment has been founded. The liver plays an important part in the destruction of toxins, the kidneys are charged with their elimination.

Eclampsia and the urea-excretion.—If we agree that the symptoms of eclampsia are due to a poison or poisons in the blood, what is it? The special constituent of urine which is most easily measured and contained in the urine in largest quantity is urea. With the acute renal disease of pregnancy the excretion of urea is greatly lessened; and with its re-establishment there goes improvement in the patient's condition. I think it can hardly be doubted that there is a relation between the phenomena of the disease and the excretion of urea.

But experiments have shown that it is not possible to produce the phenomena of eclampsia by injecting urea into the blood, and when the elimination of urea is interfered with by other forms of kidney disease, the symptoms are not those of eclampsia. Bouchard (10), moreover, found that to kill an animal with urea it was necessary that there should be in the blood nineteen times as much urea as is normally excreted in the urine during the twenty-four hours. Therefore the disease cannot be one simply of poisoning by urea.

Bouchard has isolated from the urine two substances which produce convulsions, and one which produces coma. The phenomena of eclampsia can be explained if we suppose that these are not eliminated in the urine, but remain in the blood to poison the nervous centres; and we can explain the differences between different cases, as to the number of fits and the depth and duration of the coma, by supposing differences in the proportion of these poisons.

Urea is the most powerful diuretic known. It seems probable that its function is to stimulate the kidneys to the elimination of the toxins. On this view the relation between the convulsions and coma and the diminution of urine and urea is explained; the absence of urea deprives the kidneys of the stimulus which makes them wash toxins out of the system. The degeneration of the renal cells explains the transit into the urine of much albumin.

Is the disease uræmia?—If it be admitted that the illness is the result of failure of the kidney to eliminate waste products, it may be asked, Is it not the same thing as ordinary uræmia—a condition common in both sexes, and apart from pregnancy? Puerperal eclampsia differs from ordinary uræmia in the following features:—Uræmia is a terminal stage in a long illness; and from it patients do not recover, although they may for a time get better. Eclampsia, on the other hand, often suddenly attacks women supposed to be in the best of health; and, unless it be quickly fatal, its usual termination is in perfect recovery. Uræmia does not invariably cause fits; its symptoms occur in several different groups. Sutton distinguished ten such groups. In puerperal eclampsia there is no such variety; the only differences are in the number of the fits and in the depth of the coma.

How comes it, then, that the kidneys cease their function? Three views have been taken:—

1. *The mechanical hypothesis.*—According to this, the change in the kidney is a result of the increased pressure within the belly caused by the encroachment on its space by the pregnant uterus. This is an old view, obviously suggested by the facts that there is increased pressure; that it is in the later months of pregnancy, when pressure is greatest, that the disease comes on; that the disease affects chiefly primigravidæ, in whom, from the firmness of their belly walls, the pressure is greater than in later pregnancies; and that there is an especial liability to eclampsia when, in twin pregnancy, the tension is greater than usual.

Against this are the facts that passive congestion produces a definite morbid state of the kidney, the marks of which are well known; that this particular kind of kidney, rare in pregnant women, is hardly ever found in patients who die from eclampsia; and when it is present eclampsia has been absent. The argument from the concurrence of liability to eclampsia with especially great intra-abdominal pressure is met by the fact that when the fœtus dies the kidney disease sometimes improves at once, although the pressure remains as before; that the frequency of eclampsia in pregnancies with twins is not observed in equally great distension of the belly brought about by tumours or dropsy of the amnion; and that the enormous venous congestion produced by heart or lung disease never brings about anything like eclampsia; nor can anything resembling eclampsia be produced by ligature or thrombosis of the renal veins.

2. *Nephritis.*—This is a name rather than a hypothesis. Many writers on midwifery and on renal disease have spoken of the "nephritis" of pregnancy; and many reporters of necropsies have said that the kidneys

were in a condition of "nephritis." But the latter statements have been based only on naked-eye inspection. Microscopic examination has shown that the disease is not nephritis, but an acute degeneration of the renal epithelium. Further, when patients the subject of unquestionable nephritis become pregnant, they often go through pregnancy and labour without eclampsia.

3. *The toxic hypothesis.*—Wendt has well presented the evidence for this hypothesis. On this view the disease is due to a blood poison. The accounts that I have quoted of the microscopical examination of kidneys from cases of eclampsia show microscopic appearances like those found after death known to have been caused by blood poisoning.

There are two acute diseases to which pregnant women are liable: acute atrophy of liver, and the acute degeneration of the kidney which produces eclampsia. In each of these diseases morbid changes occur both in the liver and in the kidneys. In acute atrophy of the liver it has long been known that the kidneys are always diseased like the liver, but to a less degree. In eclampsia the morbid anatomy of the liver has only recently been studied; but in the cases in which the liver has been well examined after death, changes in it, like those of acute atrophy, but less in degree, have always been found. A distinctive clinical phenomenon of acute atrophy of liver is the presence in the urine of leucin and tyrosin, in such quantities as to make their detection easy. Sir J. Williams has published two cases of eclampsia, in one of which leucin was present in the urine, and in the other both leucin and tyrosin were found when searched for by chemical methods. I know of no other recorded case in which they have been sought for in the same way. In both diseases convulsions and coma are prominent features. Both diseases are very acute. Acute atrophy of the liver runs its course within five days; eclampsia as a rule within forty-eight hours. The phenomena of acute atrophy of liver are so like those of phosphorus poisoning that some have taken all such cases to be instances of phosphorus poisoning in which the source of the poison had been overlooked.

Clifford Allbutt, in an argument in support of the toxic hypothesis of puerperal albuminuria, has drawn attention to the points of resemblance between the renal disease of pregnancy and lead poisoning. I can hardly doubt that the two diseases are closely allied. In both the extreme suddenness of their onset, often without the slightest warning, is what we should expect if a poison from without attacked the patient. If it were the case that the patient was poisoned by products formed in her own tissues, and normally excreted by the kidneys, and that the poisoning took place in consequence of the kidneys gradually finding greater and greater difficulty in getting out the toxic stuff, and at length failing in the task, we should expect the final storm of grave symptoms to be preceded by indications of gradual failure. On the hypothesis that the process is a breakdown under a load of accumulated arrears, I find it difficult to understand the onset of the disease within a few hours in an apparently healthy subject, and the rapid and complete recovery which sometimes takes place even during pregnancy and without the death of the child.

These considerations made me think it probable that acute atrophy of the liver, and the acute degeneration of the kidney that leads to eclampsia, like phosphorus poisoning, and the scarlatinal kidney, are all due to a poison or poisons from without. But this is only an opinion. If the poison exists, we know nothing about it.

A microbe has been cultivated from the urine, and has been said to be the cause of the disease. But, seeing how ubiquitous microbes are, it is not enough that a microbe should be found in the urine; we want to know the distinctive characters of this microbe, and that it occurs in eclamptic patients and no others; and these things we do not know yet. The observations and experiments that have been made are contradictory.[1]

Diagnosis.—Puerperal eclampsia has to be distinguished from—

(i.) *Epilepsy.*—In the latter disease (*a*) there is usually a history that the patient has been liable to fits; but sometimes the strain of labour will produce an epileptic fit in a patient not previously subject to them. (*b*) The quantity of urine is not diminished. (*c*) There is no albuminuria.

(ii.) *Hysterical fits.*—In these the patient does not lose consciousness. Touch her conjunctiva with a clean finger and reflex closure of the eyelids will follow. She does not bite her tongue. There is no suppression of urine; on the contrary, the fit is followed by the passage of much pale urine. There is no albuminuria.

(iii.) *Fits from organic brain disease.*—This may be old, such as a cerebral tumour or abscess. In this case there will be a history of headache, vomiting, and probably of previous fits; there will probably be optic neuritis and possibly local paralysis. It may be new, such as cerebral hæmorrhage, embolism, or meningitis. In these diseases there is not suppression of urine; if albumin be present in the urine it will not be in such quantity as to make the urine solid on boiling; and the fits will be accompanied or followed by localised paralysis.

Treatment.—We do not yet know enough about puerperal eclampsia to be able when first called to predict the course of a given case—whether the patient will get well or whether she will die. Hence when a patient, after being treated in a particular way, has recovered, we cannot surely say that the recovery was the result of the treatment. Treatment may have helped recovery, or it may have hindered it. A series of cases treated only by putting the patients under favourable general conditions would help us much; but eclampsia is a disease so fearful to the onlooker that few medical men have the courage to let patients alone who are suffering from it.

The treatment of eclampsia is therefore based, not on ascertained facts, but largely on empiricism. No treatment has been proved to do good.

I purpose first to consider the different methods of treatment that have been widely used, and to point out what I take to be the advantages and disadvantages of each. Then I shall try to point out the application of these methods to different stages of the disease.

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[1] For a summary of what has been done see Schmidt's *Jahrbuch*, Bd. ccxxxvii.

Bleeding.—This deserves first mention because it is the oldest. I suppose it began to be used at a time when it was customary to bleed for almost every disease. F. H. Ramsbotham (56) says bleeding is our "sheet anchor" in this disease; and he reflected the opinion of his time.

There are two arguments in favour of venesection—one (*a*) theoretical, the other (*b*) empirical. (*a*) The theoretical argument is that in eclampsia the pulse is generally one of high pressure, that bleeding reduces blood-pressure, and therefore is beneficial. Now, first, the pulse is not of high pressure at every stage in every case of eclampsia; therefore, assuming the foregoing argument to be correct, bleeding is not indicated in every case of eclampsia, but only in those with high blood-pressure. But, further, this hypothesis assumes that for a patient with eclampsia high arterial pressure is a bad thing, and that if the pressure can be lowered the patient's condition will be improved. Now there is not a particle of evidence that I know of to show that this is so. For aught we know to the contrary, it may be good in this disease for the arterial pressure to be high.

The empirical argument is that experience has shown that in most cases after bleeding fits do not follow one another so quickly as they did before the bleeding. Thus Charpentier, out of 210 cases treated by bleeding, found that the fits diminished in frequency or ceased in 92, were increased in frequency in spite of bleeding in 46, and were unaffected as to frequency in 72. This apparent preponderance may only have been due to the bleeding having been practised late in the course of the disease. As two-thirds, at least, of cases of eclampsia end in recovery, any treatment not injurious ought to be followed by at least 66 per cent of recoveries. This lessening in the frequency of the fits, I think, is a good reason for supposing that bleeding is at least of temporary benefit; and for adopting this measure unless it is shown to be injurious. The older authors practised very large bleeding. F. H. Ramsbotham says bleeding "should not be restrained until a sensible effect be made upon the pulse, or commencing pallor of the lips indicate approaching faintness." Such great bleeding as this is injurious, for it weakens the patient; and it therefore should not be carried so far unless the benefit is so decided as to overweigh this disadvantage.

The largest figures for comparison of the results of bleeding with those of other treatment are those of the Hôpital des Cliniques in Paris, during the years 1834 to 1872, published by Chantreuil (19), and embracing 130 cases, and of the Paris Maternité during the years 1850 to 1857, and 1864 to 1871, compiled by Tarnier, and published by Charpentier. The figures are as follows:—

[TABLE

	Bleeding.			Non-Bleeding.		
	Cases.	Deaths.	Percentage.	Cases.	Deaths.	Percentage.
Chantreuil	82	37	44	48	11	23
Charpentier	29	11	38	16	8	50
Total	111	48	43·2	64	19	29·7

There are conditions, such for instance as antiseptics and the accidents of labour, which in the cases on which these figures are founded may have influenced the results more than the treatment. Without fuller information about the cases than is given in the works from which they are quoted, I cannot assert that these figures are conclusive. They go to show that blood-letting in eclampsia is injurious and not beneficial. But we cannot be sure from them that there are no cases of eclampsia in which bleeding can possibly do good. Bleeding as a routine treatment, and in large quantities, is no longer practised in eclampsia. But in a plethoric patient, with a full hard pulse, much lividity and pulmonary congestion, I think a moderate venesection may do good by relieving the right heart.

On the toxic hypothesis of eclampsia, the temporary benefit may be explained by supposing it to be consequent on the withdrawal of part of the toxic matter from the circulation. But obviously all the toxic stuff cannot be withdrawn, and therefore bleeding can at best be but a palliative measure.

Emetics.—It has been advised to begin the treatment by an emetic, I can see no good reason why. I think it a bad practice—first, because the straining accompanying the vomiting produces venous congestion of the lungs and brain, like that caused by the fit; and, secondly, because if the patient should vomit during a fit, the vomited matters may get into the air-passages.

Purgation.—It is almost a routine practice to begin the treatment of eclampsia with a purgative, mainly, I think, because it relieves the minds of onlookers to see that something is promptly done; and a drop of croton oil is easily put on the tongue. The alleged reason is that scybala in the bowel may cause fits by reflex irritation, but I submit that this charge might more justly be brought against the purgative. I know of no other class of patients in whom scybala are held answerable for fits. A more valid but still insufficient reason is, that in mania a brisk purge often does temporary good. Another reason is that purgation used to be a routine treatment of uræmia. But the late Dr. Sutton was of opinion that purgatives in uræmia are not only useless but injurious, and I have known no one whose opinion on a therapeutic question was entitled to greater respect than that great pathologist. I know of no reason for thinking that purgation does good in eclampsia; I think it more likely to do harm; and it has the grave practical disadvantage that it interferes with the collection of the urine, and thus deprives us of the best means of prognosis. Therefore I advise against the administration of a purgative.

Chloroform.—As fits are provoked by peripheral stimuli, it would seem good practice so to treat the patient as to protect her nervous centres from such stimuli. This can be done by keeping her anæsthetised with chloroform. To do this effectively the patient must be kept under chloroform until she has been so long in quiet sleep without fits that it may be inferred that the fits have ceased. It is absurd to talk about giving chloroform when the fits come on; a fit is not preceded by warning symptoms giving sure and timely notice of its onset. When a fit comes, it is over long before the patient can be anæsthetised; besides, during the first part of the fit the chest is fixed, so that the patient cannot inhale. If the patient be comatose, chloroform is superfluous; if she be restless, it must be pushed until she is fully under its influence, and kept up. If fits do not recur, after an hour or two the drug may be withheld; and if, when this is done, the patient continues tranquilly sleeping, it may be put aside. If, on the contrary, the patient become restless, it must be resumed. So to keep a patient under chloroform requires the continual attention of a medical man for many hours together, a requirement which sometimes makes it impossible to carry out this treatment; for in country practice the doctor may be without assistance, and may have other cases to attend to of an equally serious kind, cases in which the harm from the postponement of necessary treatment may be more certain than that resulting from the withholding of chloroform in eclampsia. There is risk of death from the anæsthetic no doubt, although, as compared with that from eclampsia, it is so slight as to be unimportant.

The effect of chloroform in lowering blood-pressure is sometimes urged as a reason for its use in eclampsia. What I have said about blood-pressure under the head of bleeding applies here also.

Morphia.—Manning, in 1771, recommended opium for puerperal convulsions, but only in small doses, and combined with bleeding. Bland, in 1794, advised a grain of opium, or twenty drops of the tincture, every hour or two for three or four doses, so that, had his influence been wide, he would have been entitled to the credit of initiating the treatment of eclampsia with opiates. But his teaching was not followed. The place which morphia holds among our resources against puerperal eclampsia is chiefly due to G. Veit of Bonn.

The treatment consists in giving morphia by hypodermic injection in such large doses as to keep the patient deeply narcotised. Veit begins with half a grain, and goes on with further doses of a quarter of a grain each if necessary. Within from four to seven hours he has given as much as three grains. The object is to produce and to maintain deep sleep: so long as any restlessness continues, more morphia must be given. Veit has treated sixty cases in this way, with only two deaths. The cases in which he has been told of its failure in the hands of others are those in which the timidity of the medical attendant has prevented him from giving enough morphia. Lohlein (46) has collected from eight different German clinics 325 cases of eclampsia, which had a collective mortality of 19·38 per cent. The death-rate among those of them who were treated with morphia (number not stated) was 13·8 per cent. Cases have been reported in America successfully treated with large doses of morphia. I have used it with, I think, good results, but I know of no writer whose experience of it is so large as Veit's.

The obstacle to the general adoption of this treatment seems to be the long-accepted dogma that opium is dangerous in Bright's disease. This is a maxim of pharmacology rather than of pathology. Dr. Stephen Mackenzie has shown that morphia may sometimes be given with advantage in uræmia, and the figures of Veit certainly dispose of the maxim so far as eclampsia is concerned.

Comparing this treatment with that by chloroform, it will be evident that the advantage of chloroform is the quickness with which the patient can be got under its influence; but morphia does not require the continual presence of the doctor, and it is safer to put a patient in deep sleep with morphia than to keep up the inhalation of chloroform for many hours. The danger special to morphia is its depressing effect on respiration. This may be antagonised by atropine; moreover, danger from this cause may be reduced by keeping the patient on the side.

It is obvious that when a patient is already in deep coma the narcotic is unnecessary.

Chloral.—This treatment has been strongly advocated by Charpentier (24). He has got together statistics embracing 239 cases, and finds the mortality of cases treated by chloral alone to be only 4 per cent; that of those treated by chloral in combination with other treatment, 8·5 per cent. He remarks that possibly, as happens with most new remedies, successful cases have been published and fatal ones suppressed; but, allowing for this, he thinks the results too striking to be passed by.

As a patient with eclampsia often cannot be got to swallow, Charpentier gives the drug by the rectum. He injects a drachm dissolved in about three ounces of mucilage. If this be not retained he gives another, and, if necessary, repeats it again till it is retained. In five or six hours he gives another drachm. This is generally enough, but he has given as much as half an ounce in the twenty-four hours, and others have given even five drachms. Winckel (76) has used chloral in the same way, except that he administers chloroform until the chloral has had time to act. Out of ninety-two cases so treated he has lost only seven.

These results show a much less mortality than that which has hitherto been the death-rate of the disease collectively, namely, about 20 per cent. But the consideration which prevents me from accepting these figures, or indeed those of Veit, as representing the result of narcotic treatment, applied to all cases, is that most medical men would think it superfluous to give either chloral or morphia to a patient already comatose. Thus there would be a natural selection of the milder cases for treatment with these drugs. Comparing the results of the two drugs, it will be seen that those of morphia are slightly the better.

Pilocarpin.—Diaphoresis has long been thought good treatment in uræmia. In this condition, as H. G. Sutton put it, the "water circulation of the body" is suspended. We hope by making the patient sweat partially to re-establish this circulation; and that if we can get water out through the skin some of the toxins which are poisoning the patient may be eliminated with it. The most powerful diaphoretic we have is pilocarpin, a drug said also to reduce arterial blood-pressure. For these reasons its use in puerperal eclampsia was some years ago strongly recommended.

I think this drug dangerous in eclampsia. It makes the bronchial tubes sweat as well as the skin; it fills the air-tubes with fluid, and thus increases the tendency, great enough already, to pulmonary complications. It may even kill the patient directly by suffocation. It acts sometimes as an emetic; and this effect is bad, for reasons already stated. The value of the lowering arterial pressure I have already discussed. The published results of treatment with pilocarpin confirm these theoretical arguments. I therefore advise that pilocarpin be not given in puerperal eclampsia.

Veratrum viride has been used in America as a treatment for eclampsia. It has been used to reduce temperature, slow the pulse, and cause sweating. The dose is from 10 to 20 minims of a saturated tincture at half-hourly intervals until an effect has been produced. Its most learned and temperate advocate was the late Professor Parvin of Philadelphia. He has published a list of 284 cases treated by this drug with a death-rate of only 8 per cent. But the list is an aggregation of isolated cases and small groups of cases picked out of periodicals. Now, in matters therapeutical, successes tend to publicity, non-successes to oblivion. Hence, I doubt whether this collection of cases really represents the collective experience; it represents only the cases which practitioners have thought worth publishing.

Baths.—The necessary antecedent to getting much water out of the body is to get water in. We can get water into the body and out again through the skin by baths followed by packing. This treatment has been applied to eclampsia by Breus.

Wrap the patient in a sheet. Let her be lifted in this to the bath, which should be at a temperature of 102°, and let down into it. Let her lie in it for half an hour, the water being warmed from time to time (Breus says up to 112°, but this is an unbearable temperature for a patient fully conscious). Then let the patient be taken out, wrapped in a warm blanket, so that only her head is uncovered, and let two or three more blankets be piled on her. Then let her sweat profusely for two hours.

There are obvious limitations to this practice. It cannot be done unless there is a bath at hand, so that it is sometimes impracticable. It is troublesome. It cannot be used when labour is advanced and delivery imminent, nor when the patient is very restless. In the latter case it may be combined with the morphia treatment.

I think the principle is sound, and the results recorded from it, though few, are favourable. When a bath can be got, and labour has either not begun, or is in an early stage, or is over, I think this treatment is worth carrying out.

There are two cases in which it is especially called for. First, when a patient is dying from coma, the fits have ceased, the pulse is beginning to fail, and the temperature is normal or subnormal, its recommendation is a counsel of despair. There is nothing else we can do. The bath offers a chance of saving life. Secondly, some eclamptic patients die with a rapidly rising temperature, which may reach 108° or 109° before death. In these cases I think the cold or tepid bath offers the only hope—but a good hope—of saving the patient. Let her be put in a bath at a temperature of between 70° and 80° F., and stay in it from a quarter of an hour to half an hour, then be wrapped in blankets and allowed to sweat. The bath is here used for a double purpose, to bring down temperature and to aid elimination. I think I have seen life saved by it, for without it such cases almost invariably end in death; indeed, I think its use in such cases is imperative.

That the statistics of cases treated by baths do not show the brilliant results of chloral and morphia I take to be inevitable; for in the worst cases—those in which chloroform, chloral, and morphia are plainly useless—the bath treatment, if the appliances are at hand, can be applied more easily than in the slight cases.

Position.—This I think very important, not for any effect upon the disease, but for the prevention of the lung complications which afterwards are so dangerous. If the patient be on her back, secretions, and possibly vomited matters, will fill the air-passages. Put her on her left side, in the semi-prone position, with her left hand behind her back. In this position secretions will run out of the mouth instead of flowing back into the air-passages. Observation of the progress of labour will also be easier in this position. If the patient be narcotised she will keep this position.

Obstetrical management.—I believe that much of the mortality of puerperal eclampsia comes from the pernicious maxim, "Deliver as quickly as possible." Eclampsia depends upon a disease of the kidney peculiar to pregnancy; and for its prevention I think that labour should be induced as soon as any considerable amount of albumin is found in the urine, and other treatment has not been effective. But when the acute kidney disease (characterised by fits and urine solid with albumin) is established, the time for prevention has passed. The disease will run its course, and this course is not affected by delivery. Some cases end in recovery without delivery; others get worse after delivery. There is abundant evidence that delivery has no favourable effect on the disease. I speak of natural delivery. Forced delivery has many bad effects. Manipulations provoke fits. Dragging the child through an imperfectly dilated genital passage involves tearing of the parts. Pulling the child away when the uterus is not contracted surely leads to post-partum hæmorrhage. Cæsarean section has been proposed; and the hastening of delivery by freely cutting open the cervix, vagina, and vulva (Durhssen). Such measures have no justification unless immediate delivery greatly benefits the patient, and it does not. The right course is to let the labour go on with the least possible interference. Let the uterus do its work, and interfere only if some condition be present which makes natural delivery impossible.

I come now to the application of these means of treatment to different stages of the disease.

(i.) The patient has had a fit; she is restless and half-conscious. The aim of treatment is to prevent further fits by lessening the irritability of the nervous system. We have three agents for this purpose—chloroform, morphia, and chloral. The first two are more powerful than the third, and chloroform is the quickest, but its prolonged use is sometimes impracticable, and not free from risk. I therefore prefer morphia. Give half a grain of morphia subcutaneously. Wait to see its effect, and if in half an hour the patient be not asleep, give a quarter of a grain more, or if the patient be still very restless, half a grain. If restlessness be great, so that another fit seems imminent, give chloroform at once, and keep the patient under it until the morphia has had time to produce narcotism. Put the patient on her left side, in the semi-prone position, with her left hand behind her back.

(ii.) The patient is in coma. The coma may be simply that which follows fits. If so, it will soon be broken by the restlessness which precedes fits, and then treatment by morphia will be appropriate. It may be accompanied by re-establishment of the urinary secretion, and then it will pass into natural sleep, and recovery will follow; and, if so, no treatment is needed. It may be the deepening coma which ends in death. For such coma baths are the only remedy. If the temperature is normal or subnormal, use the hot bath, followed by packing in hot blankets. If temperature is rising, the cold bath is the only way of reducing temperature, and the only hope of saving life. Watch the progress of labour, and interfere only if conditions arise which would demand interference in a patient not the subject of eclampsia.

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G E H

CHOREA

Syn.—*St. Vitus's Dance, Chorea minor. Chorea Anglorum; Chorea vulgaris; Sydenham's Chorea.*

CHOREA is a spasmodic affection of the nervous system, characterised by involuntary movements which are sudden, jerky, and irregular; by a variable degree of muscular weakness accompanied by incoordination of voluntary movements; and by psychical disturbances, usually of the nature of hebetude and mental weakness: to these may be added phenomena indicative of arthritis and endocarditis.

No more inappropriate name could have been chosen from an etymological point of view than that of chorea,—which was applied to the disease by Sydenham,—for χόρεια means dancing, and "Chorea Sancti Viti" denoted a dancing mania prevalent in certain parts of Germany during the fourteenth and fifteenth centuries—an affection totally distinct from the disease that we have now to describe. The association of the name with the dancing mania dates from 1418, when an epidemic occurred at Strasburg, and the Magistrate sent the victims to the chapel of St. Vitus at Zabern to be calmed by religious ceremonies and the influence of the saint. As pilgrimages were made to other shrines also the epidemic became variously known as St. Anthony's dance, St. John's dance, and so on.

Sydenham's use of the name Chorea for an affection totally different from that to which it had been formerly applied, naturally led at first to a great deal of confusion; so that the use of such names as "Chorea minor," "Chorea Anglorum," and the like, were introduced to distinguish the disease now before us from the dancing mania; but as the latter outbreaks no longer occur there is now no likelihood of ambiguity. Nevertheless, even yet the name "Chorea minor" is commonly employed to distinguish the affection, as described by Sydenham, from a hysterical manifestation known as "Chorea major," which is related to the dancing epidemics of the Middle Ages.

Chorea minor, as now known to us, was first described by Sydenham in 1686, and again seven years later. Many contributions to the subject are to be found among the writings of authors between that time and the beginning of the present century; and references to their publications are made in a paper by Bernt, published in 1810; in which year another important monograph on the subject came from the pen of Bouteille in France. Since then the writings of Trousseau, Sée, Roger, and Charcot in the same country have added much to our knowledge of the subject; while similar credit is due to Romberg, Steiner, and von Ziemssen in Germany, and to Weir Mitchell, Jacobi, Putnam, Osler, and others in America; but the most important contributions to the subject have been made in this country—notably by Bright, Todd, Wilks, Fagge, Hughlings Jackson, Gowers, and many others.

Causation.—*Race.*—According to Weir Mitchell, whose information is derived from the Southern States of America, chorea is rare in negroes; and the investigations of other observers are in accord with this, for Sinkler has met with one case only in a negro; out of 554 cases Osler met with no instance of the disease in a negro child of full blood, and only four or five in mulattos; the proportion of coloured to white population in Philadelphia being as one to twenty-five. According to the same observer, at the Johns Hopkins Hospital there were 5 out of a total of 175 cases in the negro race. The disease is said to be unknown in coloured children in Cuba, although it is not uncommon among the whites. Osler's inquiries at Indian Schools, and directed to physicians practising in American and Canadian North-West Territories, have led to similar results; for, although the disease occurs in half-breeds, no instance of its occurrence in full-blooded Indians was noted.

Hereditary influences.—Though in some instances heredity plays an important part in the etiology, the proportion of cases in which chorea itself has existed in the parent is small; an indirect neuropathic heredity, in the shape of epilepsy or insanity, being more common. An interesting instance of such indirect neuropathic taint has come under my observation in a girl the subject of chorea; her father was a somnambulist, an older brother suffered from epilepsy, and a younger brother, like his father, was a somnambulist. It commonly happens that more than one member of the same family is affected with chorea. Sir W. Gowers refers to an instance in which chorea occurred in three sisters; also to several interesting associations of chorea with epilepsy, insanity, and with other cases of chorea; in one case a sister had chorea and the mother was an epileptic; in another the mother was insane, and her sister's child had chorea.

Temperament is undoubtedly one of the factors in the causation of chorea; the disease is especially apt to occur in nervous, highly strung, excitable children, while it is rarely met with in the phlegmatic.

Social status.—Such statistics as are at our disposal on this matter point to the occurrence of chorea in the lower much more frequently than in the upper classes; the percentage of the former, according to the Report of the Collective Investigation Committee of the British Medical Association, being 72·27.

Sex.—The statistics of different authors vary somewhat as regards the relative frequency of chorea in the two sexes; but all are agreed that it is more common in girls than in boys. Sir W. Gowers, from a collection of 1365 cases (100 of which were original), found the relative proportion to be almost three girls to one boy; Osler, from the records of 554 cases, found that women are affected in the proportion of rather more than two to one.

Age.—Chorea is a disease of childhood and adolescence, and it is especially prone to occur in the later years of childhood. In the Collective Investigation Committee's Report already referred to, 34 per cent of the cases occurred between five and ten years of age; 43 per cent between ten and fifteen; and 16 per cent between fifteen and twenty: but it is not stated how many of these were first attacks. Osler has analysed 522 cases as regards age, and found that 33 occurred in the first hemi-decade, 228 in the second, 212 in the third, and 62 in the fourth. The disease is rare before the age of five years; though, so far as we may judge from Osler's statistics, this does not apply so much to cases in America as in this country; but even this observer agrees that the disease is rare before the fourth year.

The cases, recorded by Richter and others, of supposed congenital origin are probably instances of a totally different affection depending on structural cerebral changes. The jerky movements which occur in such cases might easily be mistaken for those of chorea; and it is conceivable that the error might be perpetuated for some time (see "Congenital Chorea," p. 863).

On the other hand, but few cases occur after the age of twenty; according to Sir Wm. Gowers not more than 5 per cent of the total number: though a form of chorea may be met with in quite old people. To what extent we are justified in including cases occurring in the later periods of life in the same category with those which occur in childhood and adolescence is open to question, so that in this account of chorea the different varieties will be described separately.

Sex plays a part in relation to the age of incidence of the disease, which falls rather earlier in girls than in boys. According to Osler's statistics the largest number in boys is in the second hemi-decade, and the largest number in girls in the third.

Climate, Season, Locality.—Chorea does not appear to be influenced by climate; but the investigations of Morris Lewis, in Philadelphia, have been largely serviceable in establishing a distinct relation to the time of year. In an analysis of 717 separate attacks of chorea, the smallest number of cases occurred in November (24, or 3·3 per cent). A rapid rise in the number was observed in December (56, or 7·8 per cent); the number remained about the same during January and February, rose to its highest in March (101, or 14 per cent), fell somewhat in April (63, or 8·7 per cent), to rise again in May (80, or 11·1 per cent); after which time there was a steady fall to the lowest point of the curve in November.

Lewis registered the amount of sunshine and cloudy weather, relative humidity, and the mean daily barometric and thermometric readings, and concluded that weather is probably an important factor in the etiology of chorea; though he is unable to say which meteorological factor is the baneful one, the barometer and storm statistics seem to have the closest connection with the disease. Putnam, however, failed to establish a seasonal variation in Boston; and in this country season appears to have no decided effect, for of 100 cases Sir Wm.

Gowers found that 33 occurred in the first three months of the year, 25 in the second, 20 in the third, and 27 in the fourth. This observer, however, considers that a more distinct relation to season may be notable in recurrences, and quotes cases in point where, after a first attack, a recurrence took place at the same time of the year for several successive years. However, the time of the year was not constant for all the recurrences even in a given case; nor was there any particular time of the year for recurrences in all cases.

Although more cases of chorea are met with in towns than in the country, Dr. Isambard Owen's statistics show that the disease is widely distributed throughout Great Britain, and is not more apt to occur in any particular parts.

Rheumatism.—That there is a close relationship between chorea and acute articular rheumatism is now universally recognised, and some regard the former condition as a manifestation of the latter. Their association attracted the attention of earlier writers on the subject. Bouteille refers to two cases of Stahl and to two of Sauvages in which there was this association; but Hughes, G. Sée, and H. Roger did most to establish the relationship of the two diseases; indeed, the last-named observer, writing in 1866, regarded articular rheumatism, chorea, and endocarditis as different names applied to one and the same pathological condition. Other French authors have also urged the rheumatic causation of chorea; but the Germans have not attached nearly so much importance to it, except Mayer, who attributes 80 per cent of his cases of chorea to rheumatic infection. In this country, among those who early recognised the association of the two diseases, the physicians at Guy's Hospital appear to have been especially prominent. Bright, in his writings published in 1802 and subsequently to this date, clearly recognised the relationship. Babington noted the occasional association of chorea with rheumatic affections of the heart and pericardium, and was of opinion that the credit of the discovery of this association belongs to Addison.

Statistics have been collected, by various observers, bearing both upon the actual occurrence of rheumatism in the individual prior or subsequent to the attack of chorea, and upon a personal or family history of the arthritic affection. As regards the latter, Tylden found the proportion as high as 72 per cent, but the largest recorded proportion in the former category is 50 per cent. The British Medical Association Collective Investigation Committee found that rheumatism preceded the chorea in 26 per cent; in 32 per cent the rheumatism accompanied the chorea or occurred subsequently; this number rose to 46 if patients who had been the subjects of vague rheumatic pains were included.

Among the statistics collected in America with regard to this point, those of Osler, based respectively on 554 and 175 cases, gave a percentage of 15·8 among the former and 18·24 among the latter, of cases in which articular rheumatism had been present prior or subsequently to the attack of chorea. Allen Starr found, as regards actual rheumatism, that 385 cases yielded a percentage of 18; and C. W. Townsend found that in the 148 cases of his collection the percentage was 21: but by far the largest percentage is that of 54, arrived at by Crandall.

In the great majority of cases in which the two diseases are associated the arthritic manifestations precede the chorea, which occurs either as the rheumatism is subsiding, or after convalescence is established. According to Dr. Whipham, in less than 2 per cent of cases chorea precedes rheumatism; while Dr. F. E. Batten found that 11·3 per cent of chorea patients acquired rheumatism within three years, and 20 per cent within six years.

The percentage of cases preceded by rheumatism varies with the age of the patient; thus Sir Wm. Gowers met with one instance only among his cases in which there was a history of previous rheumatism in children under nine years of age; between the ages of ten and fifteen, on the other hand, there was such a history in more than one-fourth of the cases. It is obvious from this that, if attention be not paid to the ages of the patients from whom the statistics are compiled, an incorrect estimate may be formed of the frequency of the association of the two diseases. It must be remembered, however, that the manifestations of rheumatism in a child may be so slight as readily to escape detection, and that this may possibly account in part for the small number of cases in which a history of previous rheumatism can be obtained during the first decade of life. An analysis of 327 cases by Dr. Morly Fletcher shows that in 26 per cent of cases chorea is preceded by rheumatism, his figures agreeing closely with those of Dr. Stephen Mackenzie (29 per cent). Sée and Hughes both make the percentage much higher, while according to Sturges it is only 20 per cent.

Inseparable from this inquiry is the unquestionable affinity which exists between chorea and cardiac affections. Bright regarded inflammation of the pericardium as the link between rheumatism and chorea, but his views were based on an erroneous conception of the pathology of the latter disease.

Endocardial changes are common in chorea; so much so, that Fagge found such changes in 17 out of 18 necropsies of chorea at Guy's Hospital: in five of them death was accidental and due to some intercurrent affection or complication, not to the severity of the disease. The presence of such valvular changes in the heart in a case of chorea raises the question whether the endocarditis be of choreic origin, or dependent on pre-existing rheumatism. It may be impossible to answer such a question when an attack of chorea has preceded that in which the patient is seen; but, otherwise, even in the absence of a history of previous arthritic rheumatism, it may be possible to form a comparatively accurate estimate on the subject. In 40 cases of chorea with organic heart disease, Sir Wm. Gowers had strong grounds for attributing the cardiac affection to previous rheumatism- a proportion which he regards as below the actual number as he only admitted conclusive evidence. Besides the previous attack of chorea, or of rheumatism, the time that the attack of chorea, in which the heart affection is detected, has been in existence, the nature and degree of severity of the latter condition, the presence or absence of secondary changes in the cardiac walls, must all be taken into consideration.

Those observers who disbelieve the rheumatic origin of chorea insist on the fact that rheumatism is not an invariable precursor or concomitant of chorea; and there are those who, like Féré, regard the rheumatic infection only as a contingent cause, comparable to the action of other infectious diseases. Moreover, this observer does not admit the kinship of chronic rheumatism to acute articular rheumatism, but relegates that form to the category of neuropathies.

Infective diseases.—Of the various infective diseases which have been known to precede chorea, scarlet fever, which may be attended with arthritis, is the most important. So rarely are any of the infective diseases associated with chorea, however, that most observers have been led to regard them rather as indirect exciting causes of the attack. Out of 533 cases of scarlet fever, collected by Carslaw, chorea was noted in 3 only; and though Osler obtained a history of scarlet fever in 141 of his 554 cases of chorea, in not a single instance did the latter disease immediately follow the former.

Ross, however, was of opinion that chorea follows scarlet fever frequently, and thought that the association might probably be explained by the fact that rheumatism frequently follows scarlet fever.

Measles, variola, diphtheria, enteric fever, cholera, and pneumonia have all been known to be followed by chorea, and, according to Sturges, whooping-cough frequently precedes it. Another class of infective diseases, in the course of which chorea may arise, are pyæmia (with or without suppurative polyarthritis) and gonorrhœa. Straton has insisted on the possibility of micro-organisms gaining entrance through erosions in the mucous membrane of the naso-pharynx in children, and thus causing the affection. So, too, chorea has appeared in the course of secondary syphilis, which disease Féré considers has been justly regarded as a causative factor in chorea.

Instances of the association of chorea and malaria are on record, but there is no proof that the former affection is more common where the latter is prevalent than elsewhere (see "Electric Chorea," p. 864).

It is not a little interesting to note that with the onset of fever the spasms cease; a fact observed since the time of Hippocrates and frequently seen in connection with epilepsy. When one of the acute specific diseases occurs in the course of chorea, the irregular movements usually cease; according to Radcliffe, the spasms are suspended during the febrile stage, while during the cold stage they are aggravated.

Maragliano attributes chorea to the action on the nervous system of the toxins of various micro-organisms, notably the toxins of staphylococci.

Intoxications.—Closely associated with the last inquiry is that of the part played by poisons introduced into the system. The most convincing case of the kind on record is one, by Demme, in which chorea appeared after the use of iodoform in a fistula connected with caries of the cervical vertebræ; it ceased when the iodoform was suspended, and returned on resumption of the treatment.

Impoverished states of the system.—Any weakening influence seems to favour the occurrence of chorea; but most has been made of that of anæmia, as advocated by Bouchut, Trousseau, and others. The importance of an impoverished blood state as a causative factor has been insisted on more especially by Rachford. On the other hand, Osler dissents entirely from this view, though he admits that he has seen several cases in which chorea occurred in chlorotic girls at the time of puberty. Possibly the supposed etiological relationship of the two conditions is rather to be explained by the common occurrence of both during adolescence; nevertheless, Litten alludes to two cases in which chorea appeared in the course of pernicious anæmia, and in which recent endocarditis was found after death. So, too, Roeser had a case of fatal epistaxis in which similar endocarditis was found at the necropsy, and in which chorea occurred during life.

Pregnancy.—The relationship of pregnancy to chorea is very important in view of its fatality. Chorea is usually manifested during first pregnancies, and in most cases there is no other cause than the pregnancy to which it can be attributed. In other cases, however, there is a history of a previous attack of chorea, perhaps in childhood; or there may be a distant history of rheumatism; or the latter disease may immediately precede the chorea. Those who have suffered from chorea in a first pregnancy may suffer also in a second, and more rarely in a third; Féré, indeed, refers to an instance in which a woman had an attack with each of five pregnancies. If the patient escape during her first pregnancy, it is not common for chorea to occur during a second; an interesting case has, however, been recorded by Mosler in which this sequence occurred, but the woman had an attack of acute rheumatism in the interval between her two pregnancies. It is very rare for chorea to occur for the first time during a third pregnancy; indeed, no properly authenticated case is on record. Moreover, the disease scarcely ever occurs under these circumstances for the first time after the age of twenty-five. From his own cases, and some selected from those collected by Dr. Barnes, Sir Wm. Gowers found that of 28 cases 8 occurred at the age of twenty; 3 at the ages of seventeen, eighteen, and nineteen respectively; 2 at twenty-one and twenty-two; 6 at twenty-three, and 1 at twenty-four.

The onset of the chorea is most commonly between the first and the third months of pregnancy, and appears specially prone to occur at the third month. It may, however, appear at any period of pregnancy, though very rarely in the ninth month. The period of its occurrence in one pregnancy is no guide to the date of its occurrence in a subsequent pregnancy.

In a few rare cases the chorea has not appeared until after parturition; it has been known to follow an abortion.

An interesting question, which arises out of the association of pregnancy and chorea, is how much emotion has to do with the matter? A woman who discovers for the first time that she is pregnant, is more likely to be the subject of emotional excitement than one who has been in this condition before. This view finds support in Sir Samuel Wilks's observation that a large proportion of the cases occur in unmarried girls.

Emotion.—The form of emotion to which chorea has been most frequently attributed is fright; and as many mothers know this to be a recognised cause of St. Vitus's dance, probably the number of instances in which a history of fright has been obtained must be discounted. In a considerable proportion of such cases several days have elapsed between the fright and the chorea; but there are well-authenticated cases on record in which the disease followed so immediately on the emotion as to leave no reasonable room for doubting the relationship of cause and effect. One of the most striking instances of this kind is a case, recorded by Romberg, in which a little girl was attacked the same day after a severe fright caused by a dog jumping and barking at her. Von Ziemssen refers to a case in a boy where the disease became severe within a few hours after a fright; Trousseau to a case of a girl who was terrified into a nervous fit, and immediately became affected with chorea. It is rare for chorea thus to follow a fright without any interval; but among other recorded instances of the kind are two cases quoted by Sir Wm. Gowers, both in boys; in one it was the result of an unexpected pistol shot close to the ear, the other boy was caught in an apple-tree and fell in his hasty descent. On the other hand, it is rare for the interval to exceed a week, and when several weeks have intervened the efficiency of the fright becomes a matter of doubt.

Other mental emotions may likewise be responsible in some cases; notably grief, worry, or various forms of excitement: but none of these is nearly so frequently operative as is fright. So, too, mental strain, such as high pressure at school, was urged as a cause of chorea by Sturges. With the nervous system in the unstable condition of adolescence, it is not surprising that mental strain at this time should lead to a spasmodic neurosis like chorea.

Hysteria.—As chorea is so much more common in girls than in young men, and as many cases occur in adolescents, it is scarcely surprising that the two conditions should sometimes be associated in the same individual. Duchateau has collected 34 cases of the kind. This association of hysteria with some other disease of the nervous system is a matter of common occurrence, and is nowhere better illustrated than in disseminate sclerosis.

On the other hand, a few cases have been recorded in which hysteria, the great simulator, has reproduced the clinical picture of chorea with wonderful exactness; but the hysterical stigmata should serve to distinguish these cases from the genuine disease.

Imitation.—So impressed were some of the older physicians with the effect of imitation as a cause of chorea, that, according to Fagge, Addison and physicians who succeeded him at Guy's Hospital did not allow more than one or two cases of chorea to be warded with other children. It is, however, doubtful if true chorea is ever produced in this way; and not in one of Osler's 554 cases did this cause appear to have been operative. It is the hysterical variety which often arises in this way, and most of the epidemics of chorea have been made up of hysterical cases. Such epidemics have been reported, notably by Bricheteau, Steiner, Weir Mitchell, and Wichmann; but Steiner regarded the epidemic at Prague as the result of atmospheric influences, and in no way attributable to imitation. It must be borne in mind that there is a very close resemblance between some forms of hysterical chorea and the genuine disease; and, moreover, that in some of the epidemics, while the majority of the cases have been of the hysterical variety, some have undoubtedly been instances of true chorea.

Errors of refraction.—Closely associated with overwork at school is the part played in the production of chorea, in such and other cases, by eye-strain consequent on anomalies of refraction—a causal influence more especially insisted on by Stevens. De Schweinitz, who has carefully inquired into this subject, finds that there is hypermetropia, or hypermetropic astigmatism, in about 77 per cent of children affected with chorea; this proportion, however, corresponds exactly with what is usually found in childhood, for hypermetropia is present in 76 per cent of the eyes of children at the elementary schools; he concludes, therefore, that the evidence that hypermetropia is a fundamental cause of chorea is not sufficient. Osler quotes from a letter, written to him by De Schweinitz, in which the results of his more recent investigations are summarised; in it he admits that cases of chorea are benefited by the correction of refractive errors, and he supposes that in a person predisposed to chorea eye-strain may foster attacks, or even provoke them.

Reflex irritation.—Intestinal worms, gastric disturbances, dentition and the like have been regarded as causes by many, while by others their influence has been denied. They are so rarely met with that it is doubtful whether they play any part in the production of the disease; at the same time they certainly may act as proximate causes in persons predisposed to chorea.

Injury.—Blows, falls, surgical lesions, the successful and unsuccessful extraction of a tooth, and such shocks, have all been assigned as causes of chorea; but the concomitant emotion is probably the potent causative factor in such cases.

Morbid anatomy.—Macroscopical examinations of the central nervous system in uncomplicated cases of chorea are usually negative; and the same may be said of a good many microscopical examinations, though many of these have not been conducted with the necessary thoroughness. From a study of seventy-nine autopsies Raymond concludes that the condition most commonly met with is hyperæmia; the next in frequency of occurrence being softening consequent on embolic plugging of but certain nervous changes may already have taken place, in the higher centres, that had not yet invaded indicating areas; thus the appearance of the disease is sudden, though the detection of the morbid change is late. After the full recognition of the disease, we commonly learn from the friends of symptoms (now appreciated for the first time) which push back the duration of the malady. Such symptoms are the warnings, or prodroma, of general paralysis. These warnings may affect various parts of the nervous system, causing loss of power or loss of control. I will consider some of the more prominent under the heads of mental, sensory, and motor warnings.

On the *mental side* is noticed some impairment of the most specialised accomplishments of the individual; thus the musician ceases to please his trained auditors, the artist's work no longer commands a market, the artisan gets out of work, the soldier loses his good-character badge. There is, in fact, a loss of the highest power of adjustment, a kind of social ataxy which interferes with delicate social intercourse.

Loss or defect in power of attention is very common. Loss of will power, doubt and uncertainty may simulate neurasthenia; and increased irritability, change in temper, abnormal susceptibility to the influence of stimulants of all kinds are of very frequent occurrence. Hysteria and "nervousness" may also occur long before the danger is suspected. Among the *sensory warnings* we may meet with defect in smell, sudden loss of sight of a temporary kind, similar loss of hearing, temporary and local anæsthesiæ, formications, flashes of light, tinnitus aurium, and hallucinations or illusions of one or more of the senses; though in my experience these sensory warnings are not very common. Giddiness and so-called congestions of the brain may occur, or bilious attacks ending in vomiting or fainting fits. If such symptoms appear in patients who seem in other respects likely to suffer from general paralysis, they may be of value as warnings. On the *motor side* there is often well-marked restlessness, or, occasionally, stupor or undue torpor with well-marked sleepiness; slight and temporary aphasia, or loss of power of expression by speech, or by writing, or other defect of the kind, may occur, with some alteration in gait (ataxic or spastic), inequality in pupils, or change in muscular electrical reactions. There may be convulsive seizures or partial palsies, such as ptosis, external strabismus, or loss of power in one limb. Besides these local or special troubles, there is often also a general indescribable change; the person is "not himself," he is tending away from himself. There may be loss of recent memory, inability to transact business, confusion of thought, loss of animal spirits, or, on the other hand, an undue buoyancy with sleepless restlessness. I believe the character of the coming general paralysis is generally foreshadowed by the nature of these earlier warnings, though this is not always the case.

Lastly, I would call special attention to a group of warnings which might be considered with the symptoms of Social Ataxy, but as they have especially important relations I prefer to place them apart. I refer to the *moral perversions* which in not a few cases precede the recognition of general paralysis. Crimes against morality and propriety are the most common, such as criminal assaults, indecent exposure, bigamous marriages, and the like; or, again, stealing, getting money under false pretences, and reckless conduct of business, with apparent fraudulent intent, may be the kind of backsliding. Faintings, bilious attacks, and attacks of "petit" or "grand mal" also occur as warnings.

The invasion may be apparently quite sudden, and it may appear first on the motor, the sensory, or the intellectual side. The first symptoms of the disease may be but the sudden increase of any one of the warnings. A fit of one kind or another may be followed by all the mental and physical signs of general paralysis. An attack of mania, whether this be of the simple emotional or of the delirious form, may pass into general paralysis. Melancholia, or mere silliness, may be the earliest feature of the disease. In rarer cases "folie circulaire" or alternating insanity, systematised hallucinational or delusional insanity seem to initiate the disease; the fact being that the degeneration varies in the part of the brain which is chiefly and earliest affected, and consequently the earliest signs of the disease vary. To sum up: the disease may be recognised suddenly, and any form of mental, motor, or sensory disorder may appear as its first symptom. In some cases the patients seem to have been peculiar for many years, or even to have shown symptoms from early youth which, becoming exaggerated later, prove to be forerunners of general paralysis.

Forms of general paralysis.—Though various authors, including Dr. Mickle, have described certain special varieties of disease in which specific changes in the nervous centres are connected with definite mental disorders, I have not been able to satisfy myself that these divisions can be definitely made. It is convenient, however, to recognise the fact that the symptoms generally combine in one or other of the following clinical groups:—(i.) Acute or galloping general paralysis; (ii.) ordinary general paralysis, with mania and exaltation of ideas; (iii.) melancholic-hypochondriacal or stuporose general paralysis; (iv.) progressive dementia with general paralysis; (v.) general paralysis of the double form; (vi.) spinal general paralysis, that is, beginning with ataxic or spastic symptoms; (vii.) general paralysis in woman; (viii.) developmental or adolescent general paralysis; (ix.) senile general paralysis.

Forms of Insanity in General Paralysis

	Acute Mania.	Melancholic.	Delusional.	Dementia.	Total.
Bethlem	54	23	3	20	100
Virginia Water . .	39	9	16	36	100
Ticehurst . . .	19	2	...	4	25
Private	100	31	6	63	200

Note.—In the private cases the total number of patients showing degrees of Dementia was 161.

(i.) *Acute or galloping general paralysis.*—The chief characteristic of this disease is the rapidity with which the whole of the symptoms, both physical and mental, may run through their course, and end fatally. The causes of death may have been long at work: signs of mental decay, indeed, may have been passed over as unimportant till the catastrophe revealed them in their true light. The tinder was dry for the spark, and destruction is the result. In some cases death has taken place in six weeks, in others in from three to six months. In one group of such cases acute delirium or acute delirious mania ushers in the disease, and probably some of the fatal cases of delirious mania are really cases of acute general paralysis. A sudden febrile illness, a sudden moral or physical blow, or a fit may start acute general paralysis. Acute delirious mania, or a most remarkable state of restless activity, with some rise in temperature, sets in; the tremor of the muscles about the mouth is generally well marked, the defect of speech is early and well marked, emaciation rapid, face flushed, and the expression anxious; excitement is rapidly followed by mental weakness, loss of vesical and rectal control, bedsores, rapid exhaustion, and death. Fits are not, in my experience, common in this form of the disease, though they may initiate it.

It is interesting to note here that a certain number of patients who have acute delirious mania recover; certain others are left permanently weakened in mind, and a few pass from acute delirious mania into general paralysis. The general paralysis thus started by delirious mania is not always of this acute and rapid kind, but may belong to either of the other forms.

Typical case of General Paralysis.—The following is a specimen of an ordinary case of general paralysis. A married man of about 45, of active habits and with plenty of ability, not belonging to a neurotic family, who in early youth had been a free liver, fifteen years before had contracted syphilis, of which he thought very little, as he had few or no constitutional symptoms. He married when the danger of infection had passed, and for some time he indulged very freely in sexual connection; but he steadied down and was a thoroughly good and respected man of business. Certain severe money losses, due to no fault of his own, worried him, broke down his sleep and appetite, and he sought relief in alcohol. He complained of headache and loss of power, and was regarded as neurasthenic. At this time rest and travel were advised. He was very emotional, even hysterical; at other times he was apprehensive and hypochondriacal. Change and rest, however, seemed to set him up, and in two or three months he returned to work fairly well. He fell down unconscious in "epilepsy"; from this attack he recovered in a few days, but some defect remained in his speech; he hesitated, and did not articulate clearly. Now he took more prolonged rest, and when he came back to town all his friends were struck with his buoyant, exuberant health; and he said he felt better than he had felt for years. This feeling of buoyancy led to amorousness, and not content with home he got into bad company and scandal arose. He rapidly became intolerant of interference, irritable, and passionate. This state of unrest ended in a sudden outburst of mania, with violence and extravagance, during which endless telegrams and letters were sent to distinguished persons to whom he was unknown. Sent to an asylum, he fell at once into all the ways of the place, making himself the agreeable friend of one and all, and never seeming to tire in his work and play. By this time, however, the change in his expression and in his aspect was well marked: his gait was altered, his pupils unequal and sluggish, his writing changed and speech clipped. Every available piece of paper was seized for voluminous scribbling. Still little or no change was seen in his temperature or in his general circulation, though after exercise his breathing seemed to get shorter. He had lost all his finer acquirements, and though he would sing, or play on the piano, his performances were feeble and faulty. Memory was failing, but still was fair; however, he had no power of appreciating his own condition, and still thought himself in perfect health. Sleep was profound and appetite good. This period of excitement lasted for about four months, to be replaced by a phase of general dissatisfaction with his position and surroundings. He complained incessantly that he ought never to have been brought to an asylum, and he appealed so constantly for release that at last this was granted at the wish of his wife. At home he was exacting, irritable and unstable. A change into the quiet country was tried with marked gain, but a return to London was followed by another but much slighter fit which left the mind much weaker, and the patient now exhibited the characteristic "facility." He would read childish novels and cry over them, go out for the same walk daily, and be perfectly satisfied with himself and his surroundings and excursions. Sleep being very good and appetite greedy, he got very fat, his face lost expression, and his skin became sallow and greasy. This calm continued for six months, when a severe fit of an apoplectiform character left him for some days hemiplegic and aphasic. These symptoms slowly passed off, but the man now began rapidly to lose mental and bodily strength; he laughed to himself, was neglectful of cleanliness, occasionally lost control of rectum or bladder, and ate ravenously without mastication; at times he was irritable, but too weak to give much trouble. He got thinner and disposed to bedsores over the sacrum. While trying to get out of his chair he fell, and a rib was fractured, but no complaint of pain was made. Soon after other fits occurred, and for some days he was in a "status epilepticus." One arm was now noticed to be contracting, and he was too helpless to sit up.

The second and third stages had occupied a year; the bed-ridden paralytic state continued for six or seven months, during which his limbs contracted. He seemed quite unconscious of his surroundings; still the organic reflexes persisted, so that anything placed on his lips caused a swallowing movement. Thus paralysed, emaciating, and incontinent of urine and stools, recognising no one, he remained till the temperature rose, with pneumonia at the base of one lung; this rapidly spread, and with a temporary gleam of reason before his death he died quietly at the end of about three years from the time the disease was first fully recognised.

(ii.) *The ordinary maniacal general paralysis.*—It is almost always preceded by a slight period of depression, which often assumes a hypochondriacal aspect. It then resembles ordinary mania, nearly always associated with expansive benevolence with the most wonderful exaltation of ideas. The exaltation is all but universal, so that the patient believes himself to be wealthy, beautiful, a poet, an author, an athlete, and an immortal. Restlessness is also well marked in these cases, but next to expansiveness probably "facility" (as it is called by Dr. Clouston) is most characteristic. The patient, though believing himself to be so mighty, yet, like a child, is very readily diverted from one subject or purpose to another. The mania assumes the emotional or hysterical aspect, and the more marked this is the greater the chance of remission. The second and third stages are alike in all the various forms.

(iii.) *The melancholic form of general paralysis* is not always easy of detection. In many cases, as already said, there is a period of depression which may be of very short duration. General paralysis may be associated with any of the various forms of melancholia. Thus active resistive melancholia, and melancholia with stupor, may be met with. Hypochondriasis is very common in general paralysis, and I have seen melancholic patients with extreme enlargement of ideas—as, for example, to imagine such an occlusion of the bowels that if they burst—as they must—the world would be flooded by the eruption. One such patient believed himself to be a mass of syphilis, and that he would give the disease to hundreds of men in the city. In my experience hypochondriacal notions of bowel obstruction are common in the melancholic forms of general paralysis.

In many cases of melancholia there is great difficulty in getting the patient to speak or to put out his tongue, and consequently it is often hard in the earlier stages to form a diagnosis. Not infrequently I have been misled into thinking the case to be one of simple melancholia till I found the patient, though making constant complaints, was getting fat; then it was found that his pupils were unequal, his speech was defective, and his reflexes abnormal; or perhaps the onset of fits cleared up the diagnosis. Not only marked melancholic symptoms but even "persecution mania" may be an early symptom of general paralysis. I have known one man who with all the physical signs of general paralysis only exhibited simple weakness of mind and the delusion that he had scabies, which he was always anxious to show to all visitors. Another man was very dangerously suicidal because he thought he was infecting the city, and that therefore crowds were waiting to lynch him outside his house. Though suicide is not common in the ordinary general paralytics it may occur in the melancholic and persecuted patients.

It is of great importance in this group to recognise the want of proportion between the bodily and mental symptoms: thus if a man complaining of being miserable, of being obstructed in his bowels, or of being persecuted by his enemies, yet eats well, sleeps well, and gains flesh, he is either a chronic melancholic or possibly a general paralytic passing into dementia. Remissions are less common in the melancholic than in the maniacal form of general paralysis; fits of various kinds may occur, and there is generally a tendency to fatness and dementia.

(iv.) *Demented form.*—Whereas most general paralytics pass ultimately into dementia, some of them pass into weakness of mind from the outset, being from the first dull, heavy, indolent and sleepy. Although they get weaker as the disease advances, yet they rapidly fatten and are very liable to fits. In some the first stage is one rather of mental confusion than of mental defect, but dementia comes on later. Some of these demented cases begin with fits. It has been pointed out elsewhere that the progressive stages of general paralysis are summarily represented in the stages of drunkenness; for alcohol will make one man emotional, amorous, or pugnacious, another sentimental and lachrymose, and a third merely stupid.

It is common to meet with cases of general paralysis of the simple progressive demented type in general hospitals, and many such are treated at home as cases of "softening of the brain." It must not be forgotten, however, that the most placid and docile of patients may suddenly become violent and maniacal.

(v.) *Circular form of general paralysis.*—This is to my mind the most difficult of all forms to detect and to define. It is among those suffering from this form of general paralysis that so many "cures" are recorded; and it was not till I had been misled several times by such cases that the nature of the disease dawned upon me. This variety may be described in a very few words, but the description will not prevent mistakes being made over and over again. A patient (all mine have been men), of the age and with the ordinary history of general paralysis, becomes very extravagant and exalted; he will boast, rush about the town, give presents, make love, drink, and get into the hands of the police; in jail or in an asylum a diagnosis of general paralysis is made, but he slowly improves, expresses gratitude and contrition, and is sent home "well," the doctor believing he has made a mistake in diagnosis. A year later the patient is seen again, but now is in a state of profound melancholia with refusal of food, and the physician feels sure that his original diagnosis must have been wrong. Slowly this melancholia passes off again, to be followed, after an interval of varying duration, by acute mania, resembling the previous attack even in its minutest details; this gives way again to calm, and perhaps to a second "recovery." After the lapse of several years the patient, however, presents the physical and mental signs of general paralysis, of which disease he dies. I do not know any way of avoiding mistakes in these cases beyond careful observation of the physical signs.

(vi.) *Spinal general paralysis.*—General paralysis may follow symptoms of tabes dorsalis which may have lasted for some years without any apparent mental defect; or it may be preceded by marked signs of spastic paraplegia. It is possible that general paralysis may follow disseminated sclerosis; I have seen such cases, but I am in doubt about the affinity of the two diseases. It is thought by some authors that peripheral neuritis —whether due to injury, lead, or alcohol—may give rise to general paralysis; and accordingly a form of general paralysis of an ascending propagated course is described.

(vii.) *General paralysis in women.*—It used to be doubted whether general paralysis ever occurred in women, and it is certain that women of the educated classes rarely suffer from it. I think there is no longer any doubt but that even gentlewomen suffer, though to a much less degree than men. There are no special symptoms to separate the general paralysis of men from that of women. It usually occurs rather earlier in life, it has a tendency to last longer, fits are not so common, and dementia sets in earlier, the patient rapidly passing into a quiet childish state. I think the reflexes, as a rule, are much more exaggerated in women, and it is rare to meet with ataxic symptoms; this is the more interesting when we remember the rarity of tabes in women. Women may menstruate up to a very advanced stage of general paralysis; even in the second stage they may bear children, become pregnant, and be delivered normally. Children born of general paralytic parents (of either sex) will probably show signs of degeneracy; they may be idiotic, imbecile, or vicious.

(viii.) *Developmental or adolescent general paralysis* will be described hereafter by Dr. Clouston. I have seen several such cases, the majority having been taken at first to be cases of disseminated sclerosis, and it was not till later that suspicions of general paralysis arose. This form may occur in children from twelve years upwards; I see no reason why infants should not also suffer. The progress of the disease, in my experience, is not rapid; but dementia appears early, and there is rather a gradual decay than a disorder of the intellect. Paralysis soon occurs, fits are not uncommon, and the patients slowly die exhausted.

I have never yet met with such a case in which there was not evidence of parental syphilis—a very interesting fact in connection with causation of the disease in the adult.

(ix.) *Senile general paralysis.*—This variety is not recognised by many writers, and I am not inclined to regard it as a very distinct form of the disease. I have already pointed out that progressive decay of bodily and mental functions is the essence of general paralysis, and the same may be said of senile dementia; but the latter is rather a normal extinction of the higher functions, while the former is a premature and unnatural decay. It is recognised that some senile persons, mostly men, exhibit a wild, excited, and extravagant train of symptoms, during which they waste their money, go in for sexual debauches, drink, and behave like general paralytics; yet in most of these there is not the universal benevolence which is common in general paralysis, nor is there the marked defect of articulation; the physical weakness is rather paralytic from the first than mere enfeeblement. The memory, too, is more affected in dementia than in general paralysis, and if fits occur they are more probably apoplectic than epileptic. There are cases, however, in which all the symptoms of general paralysis occur in patients over sixty, and such cases must be called cases of senile general paralysis; and I know no limit to the age when true general paralysis may occur, for as age is a relative term so the diseases of old age must be relative also.

AGES OF ONSET OF GENERAL PARALYSIS

	20-30.	30-40.	40-50.	50-60.	Over 60.	Total.
Bethlem . . .	2	49	38	10	1	100
Virginia Water . .	5	37	46	8	4	100
Ticehurst . . .	4	9	7	5	...	25
Private	42	119	27	12	...	200

Duration of the disease and of its stages. This may vary from a few weeks, in the acute form, to many years in some of the chronic forms, or in those in which prolonged remissions have occurred. I have found the average duration in private cases to be about three years from the first diagnosis. Taking the history of the warnings and the early stages of the disease, I should estimate its duration in private patients of the upper class at about five years from the very beginning to the end. I have known of one or two undoubted cases which lasted from twelve to twenty years. I am inclined to think some well-marked cases never advance beyond the prodromal stage, that indeed there may be an early arrest of the process; and I believe that in occasional cases arrest may occur during either of the other stages; at any rate complete remissions may occur during the prodromal stage, the stage of excitement, or the stage of paresis. I know of no guide to the probable duration of the various stages, and I do not think there is any definite proportion between the length of the several stages. Cases which begin with very acute mania, excitement, and exaltation more frequently present remissions; cases beginning with steady loss of mental power rarely, if ever, have remissions; but the disease may be arrested. In a remission not only is there no progress of the disease, but also a gain of power; in arrest the progress is stopped but no gain is made.

Symptoms.—First I shall take those which may be called the more characteristic, and refer later to the less common symptoms. The only absolutely characteristic symptoms are progressive paralysis and progressive dementia, states which scarcely need any description. On the mental side we find loss of the highest self-control, which is shown in different ways in different persons; there is a loss or defect in the highest and most special acquirements—loss of power of self-judgment, and of judgment in affairs, often engendering extravagant notions of personal worth and power. The emotional instability is also a result of defect of control. Will-power is in defect, so that the patient is facile and easily moved; there is defect, too, in the registration of recent impressions (loss of recent memory).

On the sensory side there is frequently some loss of a special sense, and occasionally hallucinations or illusions; these again may be temporary or recurring.

On the motor side there are defects of facial expression, of speech, of writing, of gesture, and of gait.

On the nutritional side the higher or more organised tissues are replaced by fatty or degenerative products. There is also tendency to vascular and circulatory changes. The blood itself, I believe, undergoes some obscure alteration.

Loss of the highest mental control permits many impulsive acts and emotional outbreaks. An external impression may lead directly to an impulsive act or to a sudden emotional outburst—mere reflex acts uncontrolled and undirected; most of the earlier symptoms depend on this defect of control, which shows itself also in a loss of delicacy in performing the most refined and specialised acts. Tact and accomplishment are lost; the artist is no longer exact in drawing or sensitive in colour; the musician loses sense of time and harmony; the arithmetician is no longer nimble in computation; the clerk is slow and awkward in his writing; the actor loses the versatility and mobility of his features; the artisan is discharged for careless work; the domestic servant gets into disgrace for frequent breakages. The finer defects may run together in several lines: thus the clerk may write badly, speak thickly, and walk awkwardly; and the artist may be deficient not only in his own special art but in related acquirements also.

The defects in the highest control may be progressive, or they may be well marked up to a certain point and then seem to be arrested. There may be remissions also in the individual symptoms. The loss of self-knowledge is a very important symptom; for, although it is common for persons of unsound mind to fail to realise that they are unlike themselves and unlike others, this lack of self-knowledge is most marked among the general paralytics. The general paralytic seems incapable of knowing himself, and of recognising or comparing his past and present states. No amount of experience enables even a medical man to recognise the disease in himself, though he may indeed have been an asylum medical officer. Such a one may be hypochondriacal at the onset of the disease, and in dread of it; but, when he has once passed into the buoyant stage, he is quite incapable of realising his state, indeed will take a visitor round his wards and point out other patients who have similar symptoms to his own, without being able to recognise them in himself. This want of self-knowledge leads also to the grandiose ideas, the egotism, and even to the change of notions as to personality which partly depend on them. Patients may lose the feeling of self, and this may go farther, so that they have confused ideas of their own identity, and may even lose themselves altogether. I have met with patients who sought for themselves, and even took off their clothes to see if they were really there.

Megalomania.—Exaltation of ideas may depend on loss of thought-control, each idea, as it passes through the mind, being seized as real and appropriate. The mind is thus coloured by every passing wave of thought, and naturally assumes the happy guises of the great.

Exaltation, though common in general paralysis, is not invariable, nor is it confined to this disease. It may be present in ordinary and in alcoholic mania, in delusional insanity, and in paranoia; the exaltation of general paralysis differs, however, from the other forms in its universality and benevolence. The general paralytic at one moment is an actor, at another a bishop; he sees no inconsistency in being at once a god and a jockey, a poet and a millionaire; as a rule he is benevolent in his exaltation, being willing to grant power and wealth to those about him. The patient with monomania of grandeur as a part of delusional insanity is usually content with his one great idea, and is not in the least disposed to give up his power or to share it with others.

Exaltation is not only associated with the maniacal or expansive form of general paralysis, but may occur also in the other forms of mental disorder which may form part of it. I have met with melancholic general paralytics who have thought that millions of devils were torturing them; and I may refer again to more than one patient who supposed his bowels to be obstructed by millions of tons of fæces. In other cases delusions with ideas of persecution have been associated with notions that all the world was in league against them.

It is noteworthy that in general paralysis there may be a dual consciousness, so that while the patient talks of his millions of gold and diamonds he may yet remember the state of his balance at the bank, and act consistently therewith.

Micromania is a name used in contrast to megalomania and to indicate what Dr. Mickle calls "belittlement." This occurs more frequently in general paralysis than in any other form of mental disorder; but it is rare even in this. The patients say they feel so small they can go under doors, or they can get through cracks and keyholes. This feeling of belittlement may be associated with some cutaneous anæsthesia; as the opposite idea of greatness or swelling of body may depend on hyperæsthesia. Micromania is met with in certain other cases, but chiefly in senile degeneration, or mental degeneration of one kind or another. I have met with senile patients who spoke of themselves, and of all about them, as "little things."

Emotional instability is very rarely absent in general paralysis, and is often one of the earliest symptoms; there is a tendency to exaggerate both the joys and the sorrows of life; usually it is marked by increased irritability, so that the formerly docile, good-tempered man becomes hard to live with. In rare instances the change is of the opposite kind, the bad-tempered man becoming more easy to manage. Crying and passion are both common in general paralysis, and it is well to remember that hysteria occurring in a middle-aged man is very frequently an early symptom of general paralysis.

The general paralytic is pleased with a feather and tickled with a straw; he will sob over the penny novel and weep over the vulgarest drama. The ease with which men are affected by emotional causes is a gauge to their stability, and is a good indication of the decay produced by age or disease.

The defect of will-power, already described as one of the well-marked features of the disease, and the *facility* (of Clouston) are seen not only in emotional weakness but in the want of will which makes it often easy to guide these patients for their welfare; determined and obstinate perhaps if argued with, they may be diverted by changing the direction of their thoughts. Want of will may show itself in some cases as true *folie de doute.*

Memory is affected in very various ways: one of the earliest complaints made by patients themselves is that their memory is not what it was; but, as this statement is made by most men over forty-five, it does not attract much attention. Often the memory seems to be more seriously affected at first than later, probably because fewer calls are made upon it; the loss of memory is slow and progressive, in some cases the defects suddenly follow fits, which may leave a distinct gap. It begins with failure in the more recent and more isolated facts; then it leads to a strange jumble of ideas and a mixing up of the past with the present—the real with the ideal. The memory may seem to recover itself during remissions. Generally speaking, the loss of memory is not nearly so well marked as in alcoholic paralysis.

On the *sensory side* there is usually loss of acuity in one or more of the senses, but it is not common in general paralysis to come across cases of progressive failure ending in complete blindness or deafness; though I have often met with cases of more or less complete blindness, or deafness of one ear, which has proved transitory. There is no special change in the discs associated with the disease; in some cases there is an excess of vascularity in the early stages, in others atrophy; and in some the vessels seem to be placed in channels which have rather dark lines along their edges. Sometimes, towards the close of the disease, there is marked optic degeneration; at others temporary or permanent blindness will affect one or both eyes. It is probable that in the optic nerves, and in other peripheral nerves, degenerative changes generally take place if the patient live sufficiently long.

The pupils are generally unequal; the inequality varying, one pupil being larger one day, the other another. The pupils are frequently irregular in outline, but the most important point, in my opinion, is to ascertain whether they react to light and accommodation. We often meet with ataxic symptoms in which some reaction to light or accommodation is retained, though this is much less than normal; and there is little proportion between the spinal and the pupillary symptoms. It is common, however, to meet with small pupils in cases which began with locomotor ataxy; and it is also common to see dilated pupils in rapid cases, and in the later stages of the disease. There is no direct relation between the mental symptoms and the dilatation of the pupils; nor is there any direct connection between the dilatation of the pupil on (say) the left side and the exaltation of ideas. The pupils, then, are generally abnormal, they vary from time to time, they generally react defectively to both forms of stimulation, and they are often irregular and eccentric in form.

It has been taught in France that in general paralysis there is loss of power to detect pepper. I have tried to find this symptom but have failed, and I do not attribute any value to it. Many cases of hyperæsthesia have been described, and I have met with a few. In some there is only a local sensitiveness, which in most cases passes off; in other cases patches of anæsthesia occur, as I shall state presently, but as yet we can attach no special value to these symptoms.

Headache or head tenderness, neuralgia, and other nerve pains have been described by Batty Tuke and others; but it is not a common experience for me to meet with these.

Hallucinations and illusions of the various senses may be present at any time of the earlier stages of the disease. In my experience they are not so common as in other forms of mental disorder; but it is a mistake to think that they are rare. Dr. Mickle says that more than half his patients had hallucinations during one period or another. Those of sight and hearing were the most frequent, and were about equally common. Many general paralytics say they see and hear things, as the outcome of their boastful delusions; they think they are God's messengers, and must therefore hear from Him. There is nothing special in the character of the hallucinations, which may be single or multiple, pleasant or painful, constant or occasional. In some few of my own cases the hallucinations followed the same course as in systematised delusional insanity, and were associated with ideas of persecution, of spies or detectives, of whisperings and annoyance.

Common sensibility may be reduced or almost annihilated; I have known patients rub holes in their bodies or their limbs; and one case of a man who slept placidly while his hand was being roasted. It would seem that there may also be deficient reflex sensibility, so that in them there is an especial danger of choking by the food in the throat, which is not duly stimulated to swallow.

The *disorders of muscular or motor control* are many and characteristic; they are of two classes: the passive, as seen in defective reaction and defective expression; and the active, as seen in disorders of articulation, writing, and gesture. The face wears a mask, the cheeks are flabby without lines or folds; in some cases the patients look as if they had blown out their cheeks, in others the frontal muscles have to be fixed before the rest of the face can be moved. Besides loss of expression there is loss of control, as seen in the very tremulous over-action or ill-directed action. In many cases in which the knee-jerks are exaggerated there is also great excitability about the facial muscles; the tremor is usually most pronounced about the muscles of the lips, the tongue also being very greatly affected; for a time indeed it is ataxic, so that its movements are irregular and awkward. There may or may not be fibrillar tremor, but I do not think this is very common. The tongue from being ataxic becomes more paralysed and rests on the teeth, taking an impression from them along its sides. Certain words are not readily pronounced. I believe it will be found that some general paralytics fail in the labials, others in sibilants or dentals. Tremor of tongue and difficulty in speech are greatest after mental or moral excitement, and after eating. There is often loss of control over the cheeks and lips, so that saliva flows over the chin, or food collects about the gums.

Various forms of disorder are associated with the function of mastication; thus there is often a tendency to smack the lips, or to movements like sucking, grinding of teeth, or swallowing. The grinding of the teeth may go so far as to fracture them.

The *handwriting* in general paralysis is almost pathognomonic. In the earliest stage the patient often writes voluminously in a rather shaky hand, and it is noteworthy that the words are often left incomplete; syllables are omitted and the letters are not attached one to another. In some cases, however, the handwriting improves in legibility, for the patient, finding his inability to write quickly, begins to write large text like a child; yet even then the letters are often separate, and the words shortened. Although the writing has much the character of that of an old man, in this latter case the shakiness is not associated with the separation of the letters. In alcoholism, however, the shakiness and the elision or dropping of letters may occur together.

Muscular defect in gesture is seen in the orator and the actor, and may give way to ludicrous exaggeration of the natural expression. The gait varies greatly, there being many grades between the extreme ataxic and extreme spastic walk. In some cases the symptoms may vary, so that a patient who at one time seems hopelessly ataxic, at another may lose this weakness. In many cases the symptoms differ on the two sides. There is very often a great restlessness, which on the muscular side is seen in incessant movement or perpetual writing of letters.

Knee-jerks may be absent, normal, or exaggerated; or they may differ on the two sides: they may also vary from time to time.

Fits and Palsies.—During the progress of general paralysis fits occur commonly, but by no means universally. These may be the earliest symptom, though more frequently they occur in the later stages of the disease. The attacks have been called, according to their severity, congestive, epileptiform, or apoplectiform seizures.

I have already pointed out that the occurrence of hysterical fits in middle-aged men is of serious import; I need only add here that it is not uncommon to meet with cases in which hysteria was the first symptom of general paralysis. A hard-worked man who has had a great deal of worry and anxiety, when consulting the doctor, bursts into tears, sobs, then laughs, and for a moment cannot speak. Such emotional instability may precede the other symptoms by a year or more.

A slight attack of giddiness, some temporary loss of consciousness similar to that of petit mal, sudden transient loss of speech, blindness, or deafness may mark the onset of the disease; such symptoms pass off and recur. They may be present, under stress of mind or body, at irregular intervals several years before the other and more easily recognised symptoms of general paralysis are noticed.

Congestive or other seizures may start the active mental disorder: thus directly after a fit a man may show great mental excitement; but in the great majority of cases the fits first appear after the patient has passed into the second stage; so that some have spoken of this as the "fat, fatuous, and fitty stage." It is common experience that patients grow fat before the onset of the fits. There may be slight warnings, resembling attacks of petit mal, before severe apoplectiform fits occur; but this is by no means constant, as the first fit may be very severe, or even fatal. It is common to meet with some gastric disorder immediately before the fit, and increase of temperature is not an unusual warning of this danger. The fits are very irregular in their recurrence; and though for some time, affecting the same centres as epileptic attacks, they may present similar features, yet it is common to meet with fits which do not affect the same parts in their recurrence. The seizures may continue for days together, the patient remaining in a *status epilepticus.* Fits are most common toward the end of the disease, and are often the immediate cause of death. As a rule, marked mental degradation follows the onset of the fits, but in a few cases I have met with temporary improvement after epileptiform seizures.

Though the seizures may be most marked on the sensory, motor, or vaso-motor (or "organic") side, yet, as a rule, in complete fits all these parts are affected. I have seen several cases in which temporary loss of sight or of hearing, generally on one side, has preceded other and graver symptoms; in others local or general hyperæsthesia was present. On the motor side sudden loss of power in one hand is the most common failure, though occasionally speech may be embarrassed while consciousness remains perfect. I think very gravely of any temporary loss of power occurring in middle-aged active men.

It is not uncommon to meet with so-called "bilious attacks," which may have a vaso-motor origin.

The fits vary greatly in form and in degree. I have already spoken of the slight passing attacks which occur most frequently in the earlier stages.

Next in order are the epileptiform fits, which cannot by inspection be distinguished from true epilepsy. They are most commonly associated with complete loss of consciousness, though I have seen a patient convulsed while at the same time he was conscious, and was swearing at his disorderly limbs.

As in epilepsy, these fits frequently start in a definite spot and follow a definite line of development; but this is not always the case. The epileptiform fits may pass off rapidly, leaving the patient tired, but ignorant of what has happened; but, as a rule, the epileptiform fits of general paralysis leave more physical and mental weakness behind them than a similar nervous discharge in epilepsy would do.

While the result of an epileptiform seizure is more marked in general paralytics than is a similar fit in an ordinary epileptic, it will be seen, on the other hand, that the apoplectiform seizures in them are followed by less marked and less permanent disorder than is a true apoplectic fit.

I need not describe the character of the epileptiform fits themselves, as they start from the same centres as do those of true epilepsy, and generally run a similar course. In the apoplectiform fits we have usually one-sided convulsions, the fits often starting with a distinct deviation of the head and eyes to one side, and with rigidity and convulsive movements. These tonic and clonic convulsions may recur for hours or days together, as a result of external stimulation, or spontaneously. During or after the convulsive seizure there is most profuse sweating; and there may be involuntary actions of bowels and bladder. The convulsions may be so severe as to fracture the limbs or the teeth. There is frequently pallor at the onset, but this is replaced by congestion of the face and neck, which may pass into lividity. The pupils may be equal but are commonly dilated; there is complete insensitiveness of the conjunctiva.

The fits of this description may leave a patient comatose, or he may remain for hours in a very unstable state; so that the slightest external stimulus will lead to a fresh outburst of convulsions. As a rule, the hemiplegia which follows such apoplectiform seizures resembles in all details that of true apoplexy; thus with right-sided hemiplegia aphasia will probably occur. This hemiplegia, however, is of comparatively short duration, passing off much more quickly, as a rule, than that following a coarser brain-lesion. I have seen an apoplectiform seizure, leaving temporary paralysis, followed by a seizure of the opposite side, associated with recovery of power in the side first affected. After a severe apoplectiform fit there may be coma, followed by maniacal excitement; which, passing off, leaves the patient hemiplegic. In some patients such fits recur for many months or even for a few years; but, as a rule, frequent fits mean speedy exhaustion and death.

In all cases of fits the treatment must be purely expectant: bleeding or severe purgation is not to be thought of; chloral hydrate, in 20-grain doses, administered by enema, has seemed in some cases to shorten the fit and reduce the excitability. During the attack the patient should have his clothes loosened, and he should be kept from all sensory stimulation as much as possible. Unless very weak, it is well not to trouble about feeding by the mouth for a while, for the attempt will probably give rise to new convulsions. I do not think any good is to be derived from sinapisms or the like. In some of my cases amyl nitrite was given at onset of the fits, but I cannot report any favourable result from it. Under the head of apoplectic seizures some observers class the paralytic seizures, which may come on during general paralysis, in which a limb or part of the body may suddenly lose power for a time without any convulsions. These cases resemble apoplexies due to brain-softening.

The paralysis generally increases, and may become associated with contraction of the limbs, even to an extreme degree. The contraction may have a hemiplegic or a paraplegic distribution. With great amount of disablement muscular strength may be maintained. I have met with men who could register a very high power on the dynamometer, yet who could hardly direct a pen.

In some of the cases of general paralysis with ataxic symptoms I have met with various changes in the skin. In one case the joints were affected by Charcot's disease, and symmetrical bullæ formed about the feet; there was also a marked and persistent *cutis anserina* and an abnormal growth of hair on the legs. I know of no special odour associated with the disease. Herpetic eruptions may occur, more particularly in the earlier stages of the disease. Carbuncles may play an important part in general paralysis; they may usher in the disease or they may bring it to an end; again, I have seen very prolonged remissions follow serious carbuncles. There is nothing characteristic about the temperature of general paralysis; many observations have been taken, and these show that in some cases the disease is associated with a slight persistent rise in temperature, this rise being higher, as a rule, at night. During the middle and part of the later stages the temperature is often normal or subnormal; and I have known a chronic patient have a subnormal temperature for many months. Towards the end, when there are secondary complications, the temperature varies greatly and may be very important as a warning of such secondary disease—of pneumonia, for example. Before the onset of convulsions, and during and after these, a rapid and marked rise may occur. The rise is associated with or followed by profuse sweating.

General Nutrition.—Usually with the early stages of the disease there is a tendency to waste; later, to grow fat, although afterwards the fat is reabsorbed and wasting sets in again. There is still a great tendency to hæmatoma. Brittleness of bones and defective power of resistance are common, but at the same time the power of repair of injuries is often maintained at a high standard. I have no doubt that in some advanced cases marked arterial changes occur; but I cannot find any evidence of early or widespread arterio-capillary fibrosis: nor do I find grounds for believing that changes similar to those met with in Bright's disease are common. Yet I have often found evidence of syphilitic changes in the arteries of the brain. There is a liability to subcutaneous hæmorrhage; hæmatoma and pachymeningitis being among the results.

Capillary congestions are frequently seen over the malar bones; and in some of the hollow viscera—such as the bladder—they may lead to hæmorrhage. There is some change in the blood itself; but, notwithstanding the observations of foreign observers, I think nothing characteristic has been discovered in it. The skin becomes sallow and waxy, erythematous disorders occasionally appear, and if the nursing be inefficient bedsores will form. There is a great tendency to profuse sweating after fits, and this may lead to sudaminal rashes. "Tache cérébrale" does not seem to me to be of any value as a symptom, at any stage of the disease; it is common to many and various diseases, and may indeed be found in the normal state.

The *pulse* in general paralysis varies greatly: not infrequently in the earlier stage it is above the normal in tension and number; it soon loses power, often remains rapid, small and compressible, and may become very slow, when it points to increasing weakness of the heart. The pulse rises, as a rule, with the temperature. There is no characteristic sphygmographic tracing of general paralysis.

Respiration varies, as a rule, with the pulse. After fits it is common to meet with Cheyne-Stokes breathing, which may be of short duration or may persist; in a few cases respiration becomes very slow. Sleep in the first stage of general paralysis is often good, but the patient does not appear to need so much as usual; he will sleep profoundly, wake early, and begin his restless day betimes. In some cases this will lead the patient to the extravagant notion that he has discovered the root of all evil to be too long a rest in bed. Sleepiness is, however, no uncommon symptom of the earliest stage; patients may sleep almost as soon as they sit down; they sleep over a meal, and sleep well at night also. One teacher has gone so far as to assert that sleeping after meals is a cause of general paralysis; it is certainly a common symptom of it. In the second stage sleep is generally more normal; but towards the end there is restlessness, associated with the bodily discomfort of incontinence of urine and, in neglected cases, of bedsores.

The *urine* of the general paralytic presents no constant abnormal quality; I have met with sugar, and also with albumin, and not infrequently I have noted the high specific gravity, with abundance of lithates, which corresponds with the rapid tissue-changes of the body. Moreover urea and uric acid in excess, or sulphates in excess, are common. Occasionally, after very severe convulsions, I have met with albumin in the urine.

Remissions of the symptoms, both mental and physical, may occur in most of the periods of the disease. These are most common in the earlier stages, and they are more frequently met with in patients who have begun with symptoms of maniacal excitement. In part this is due to the fact that such patients have accentuated their symptoms by giving way to alcoholic and other excesses. The remissions may be complete or partial; thus, as I said, both mental and physical symptoms may disappear for a time, but it is much more common to meet with cases in which the mental symptoms subside while the physical ones are progressing.

I have met with one patient who, while excited, showed little or no ataxy in his gait, but who, as soon as his mind was more clear, was quite unable to stand alone; in this case there were several remissions, each associated with similar symptoms. I think that the symptoms which are most persistent are those affecting the finer adjustments, such as speech and writing. During the remissions the patients may resume their professional work for a time, and may rejoin their families; though, as a rule, the return to old ways leads to rapid recurrence of the symptoms. It is rare to meet with patients who have had more than one complete remission. Acute stages may be followed by intervals of quiescence, and these again may be followed by excitement; but after each period of excitement there is a marked degradation in the patient's state of mind. These cases with recurring attacks resemble in many ways the general paralysis of the double form.

Arrests, if not remissions, may occur even when the patient has passed into the paretic stage; and, in one case at least, I have seen the disease arrested for many years after the symptoms had gone so far as to lead me to think the patient was dying of fits in the last stage. Remissions may follow injuries or acute diseases, such as pneumonia: or they may follow suppuration, or some skin affection.

Terminations of general paralysis may be natural or accidental. The natural end depends on the progressive disease affecting the whole system; thus exhaustion and slow death by heart or lungs may occur; or the exhaustion may depend on inability to assimilate proper food: besides the weakness there may be diarrhœa, bedsores, and vesical catarrh. Implication of the bulb may lead to irregular breathing, and to defect of power in respiration; and this may cause clogging of the lungs, pneumonia, and death. Phthisis, or at any rate degenerative lung disease, may end the case. Whether the phthisis be more often fibroid or not I cannot say. The fits of one kind or another, which may come on at any stage, may cause death either directly by their frequency, or be associated with some brain lesion or blood lesion, such as hæmorrhage, which may lead to death. Such are the terminations which I have called natural.

The accidental terminations of general paralysis may come by fracture of ribs leading to pleurisy or pneumonia; and by fractures of other bones leading to other complications. Bruising may lead to hæmatoma, which may exhaust the strength, or give rise to blood poisoning or to abscesses. Local hæmorrhages may follow slight bruises on the surface, or be shed into the membranes of the brain. Exposure to heat or cold may cause death. Impaction of food in the air-passages, or in the œsophagus, or the inhalation of fluid during a fit, or as a result of paralysis about the throat, may cause immediate or more distant death; suffocation may occur during a fit, if the patient turn on his face. Suicide may be the result of such a delusion as that the patient can fly; or it may be the result of mental depression.

Pathological anatomy.—In considering the morbid appearances of the nervous system in general paralysis we note at the outset that there are none, so far as our present knowledge goes, which can be regarded as peculiar to this disease. In the very great majority of cases, however, which have been regarded clinically as general paralysis, the autopsy reveals a complex of morbid conditions which, whilst perhaps scarcely so suggestive as the group of symptoms which go to make up the clinical picture, is nevertheless sufficiently characteristic to bear out the diagnosis founded upon the latter. As regards the brain, it may be affirmed that certain morbid conditions are found there in a more pronounced form in general paralysis than in any other disease in which this organ is involved.

The naked-eye appearances of the cerebro-spinal axis, and its enveloping parts, with which we are familiar at the autopsy of a case of general paralysis in which the disease has lasted the ordinary period, are as follows:—The skull-cap (sawn through at a standard level) is commonly heavier than normal. In some 25 per cent. of cases hyperostosis is noted. The diploe is obliterated or deficient in about three-fourths of the cases; the sutures are more or less obliterated, or covered with calcareous deposit, and exostoses occur. The dura mater exhibits changes in about one-half of the cases, is then thickened, congested, and adherent to the skull-cap, especially along the sagittal and coronal sutures and over the frontal bone. The Pacchionian bodies are unusually prominent, causing marked pits in the bone above them. Calcareous deposit occurs in the falx cerebri. The inner aspect of the dura shows rusty discoloration, or localised blood-clot, which may be surrounded by membrane; or cystic formations may be present ("arachnoid cyst"); or membrane of various degrees of consistence and extent may be stripped off, especially from the most prominent part of the convexity of one or both hemispheres, or the temporal fossæ.

The serous fluid in the subdural and subarachnoid spaces and in the ventricles is turbid and increased in amount, 4 to 6 ounces escaping. The arachnoid, where it bridges over sulci, and over the space between the crura cerebri and optic tracts, is thickened and milky, or opaque and swollen, watery or gelatinous; and the leptomeninges generally are thickened, œdematous, hyperæmic, and show milky streaks along the course of the main veins; these conditions being most marked over the convexity of the parietal and frontal lobes. The frontal lobes frequently adhere to each other, the adhesions being easily broken down. The pia, on stripping, is found to adhere to the cerebral cortex along the summits of the gyri, though for short distances only, and at this stage not strongly. Indeed, at the usual date of death there is frequently no adhesion, the leptomeninges stripping, on the contrary, with undue ease.

Adhesions are commonly present over the frontal and parietal lobes, at their convexity; and are especially noted over the central gyri and the bases of the frontal gyri. The occipital lobe does not usually present adhesions. After removal of the pia the cortex at the site of adhesion presents a worm-eaten appearance.

Summing up in respect to the meninges—there is evidence of diffuse, chronic meningitis in over 80 per cent of the cases.

Of the brain there is a general atrophy: the gyri are shrunken, the sulci and lateral ventricles are widened, and there is a loss of weight (1250-1270 grams would represent the weight commonly found). The organ is flaccid; it collapses when placed upon a table, the hemispheres separating posteriorly, and the posterior extremity of the corpus callosum becoming torn—an indication of softening. Atrophy, though general, is especially noted at certain parts, such as the central and precentral gyri, the basal ganglia, the pons, and medulla. Softening is also general, with foci of special intensity; it occurs irregularly in the gray and white matter, especially in the parietal, central, and temporal gyri, in the island of Reil, in the external and internal capsule, and the lenticular nucleus; it is also met with in the basal ganglia. The focal softening is ascribed to blockage of minute arteries. There is often considerable atheroma of the main arterial trunks, and hæmorrhagic foci exist, especially in the centrum ovale, the basal ganglia, and the pons and medulla; these are ascribed to degeneration of the walls of the arterioles in conjunction with increase of the arterial blood-pressure. Frequently also there are traces of former hæmorrhages, in the form of cysts and scars. The cerebellum participates in the above degenerative changes, but not in a marked degree: here softening—general and local—and hyperæmia are the most marked naked-eye alterations.

The cortex cerebri exhibits foci of softening, general hyperæmia, or patchy discoloration. It is also atrophied, especially in the frontal and parietal regions. In the white matter similar conditions exist; and the perivascular spaces are dilated. The ventricles are dilated, and the ependyma of the fourth ventricle is congested, thickened, softened, sometimes gelatinous, sometimes tough; its surface is granular in varying degree. The same conditions of ependyma are found in a lesser degree in the other ventricles.

The cranial nerve-roots show gray degeneration and atrophy: these conditions have been noted in the olfactory bulbs and tracts, the optic tracts, the third, fifth, sixth, and seventh nerves.

In contradistinction to some of the conditions noted above we find exceptional states, chiefly affecting the coverings of the brain: of such is atrophy of the skull-cap, which is therefore lighter than normal; the dura mater may appear quite normal—this is less unusual than might at first be supposed,—or there may be evidence of pachymeningitis externa, and rusty deposits may be present on the outer aspect of the dura; granulations occasionally occur on the outer aspect of the arachnoid, and adhesions between dura and arachnoid. Adhesions between the pia and cortex may be very rare, or unusually situated, or absent; even though the case be not an advanced one. The ependyma may appear normal, or at most slightly granular; in about 20 per cent of the cases naked-eye evidence of disease in this structure is but slight.

Turning to the spinal cord and its investing membranes: there is naked-eye evidence of disease in the membranes in about 39 per cent of the cases. Adhesions exist between the vertebræ and dura mater; and the cord-tunics are thickened, opaque and hyperæmic, chiefly posteriorly. There may be blood-extravasation upon the inner aspect of the dura; but deposits, whether hæmorrhagic or calcareous, are much rarer upon the cord-membranes than upon those of the brain. Occasionally there are adhesions between the dura and the pia. The cord itself is chiefly softened, but sometimes indurated. The different columns, chiefly the lateral and posterior, may show gray degeneration; there is evidence in some cases of disseminate sclerosis. Lesions of the columns, manifest to the naked eye, are less commonly observed in general paralysis than in the ordinary degenerations of these parts. In some cases the entire cord is diminished in size; or there is diffuse softening. The cord-changes, as will be seen when histological conditions are referred to, are very various.

In acute cases, and in those which succumb in the earlier stages of the disorder, the naked-eye appearances, as might be expected, are less indicative of disintegration and degeneration; in place of these are exhibited the signs of acute disease:—the blood-vessels are everywhere congested, whether in the diploe, dura mater, leptomeninges, or brain-substance; flakes of lymph occur in the cerebro-spinal fluid, and there may even be, at points, pus-formation—purulent meningitis (Kaes). The membranes are tense, and on cutting them the full-volumed vascular brain projects, and its diminished consistence is noted. Adhesions between the pia and cortex are strong and extensive. Such adhesions are present in at least 70 per cent of autopsies of general paralysis. There may be recent blood-deposit in the subdural space.

Histological changes.—The Brain.—Seeing that when the disease has lasted its customary period, end-products only, of no particular importance, are met with in the brain, cases are selected, for the purpose of histological examination, in which death had taken place in an earlier stage; preferably those in which the duration of the disease has not exceeded one year. In such a case we find in the cerebral pia mater that the vessels are prominent, with thickened walls and abundance of nuclei. There is increase of the cell-nuclei of the sheath of the vessels which pass from pia to cortex, with thickening of the sheath. The septa passing from pia to cortex are thickened and increased. There is free exudation into the meshes of the pia. The trabeculæ between arachnoid and pia, as seen in a sulcus, are bathed in inflammatory exudate. The various structures of the cerebral cortex give indications of disease. Thus the blood-vessels are exceedingly numerous, distended with corpuscles, and tortuous; at points along their course they exhibit dilatations, at which points the lumen, on cross-section, is found occluded by a yellow mass, which takes aniline blue stains very deeply. The nuclei of the vessel-wall are greatly increased, and extravasated leucocytes are apparent. The coats of the vessels are thickened, and there is proliferation of the endothelial nuclei. Hyaline degeneration, or fatty change, of the vessel-wall may be present. Hæmatoidine crystals about the vessels point to extravasated blood. In connection with the increase of nuclei about the vessels and in the neuroglia it is to be observed that the shrinkage and disappearance of the tissues may be held to account for some of the apparent increase. The vascular lymph-spaces, subadventitial and perivascular, show collections of lymph-corpuscles and blockage. The perivascular sheaths become much distorted by the contraction of the vascular processes of the spider-cells.

The supporting structure (neuroglia) exhibits deep staining in many parts of a section; this is especially marked in preparations by the fresh method of Bevan Lewis. Instead of a ground-substance scarcely stained, there is found a mottled, patchy-blue staining between the nerve-cells, a granular or fibrillar condition. In other parts the staining is defective. With these conditions deeply-stained round bodies, apparently nuclei, are found in abundance; and glia-cells ("spider," "lymph-connective") are numerous and prominent, with large, deeply-stained bodies and numerous processes.

These spider-cells are especially noteworthy in the outermost layer of the cortex, in contiguity with the apical processes of the pyramidal nerve-cells, where their increase is earliest noted; and also in connection with the vascular sheaths. A strong "vascular" process passes from the cell to the vessel-wall, ending there in an enlarged extremity, a nucleated mass of protoplasm; and from the body of the cell other processes radiate. But these cells form also prominent objects between and around the nerve-cells; and it is noteworthy that their processes may be traced to the degenerate bodies and processes of the nerve-cells, and are found surrounding the latter. These conditions are best shown by the fresh method above mentioned. At a later stage the spider-cells are transformed into a fibrillar meshwork.

The nerve-cells and their processes exhibit degeneration: the latter are stunted, granular, and blurred in outline; later they are thin, with broken or irregular course, disintegrated, or destroyed. The apical process suffers early, a point to be noticed in connection with the great development of spider-cells in the outermost layer. The cell-bodies exhibit various changes; their contour is blurred, they stain irregularly, and show granules or fuscous change (formation of brownish-yellow pigment), sclerosis, and occasionally vacuolation. The nucleus is no longer distinct. The cell-shape is altered: it is swollen, irregular, locally defective; later it is atrophied, or merely a broken-down residue of pigment. There is, in the result, considerable destruction of the cells. In the smaller cells the nuclei appear swollen. The cell-nuclei in many cases show altered staining reactions, the reasons for which are obscure.

The nerve-fibres of the cortex also show changes; alike in the outermost layer (parallel to the surface) and in the intracortical radiations there is degeneration (wasting, disappearance) of the fine medullated fibres Tuczek). Some observers assert that this is especially the case in the frontal lobes, and in the next place is most frequent in the parietal and temporal lobes, and the paracentral gyri.

Ljubimow has described the degeneration, atrophy, and—in the later stages—disappearance of the "association-fibres" of the cortex: this degeneration proceeding from before backwards over the cortex. This nerve-fibre degeneration is regarded as a primary degeneration, independent of any inflammatory process. Colloid bodies are seen, especially in the outermost layer, which are ascribed to degeneration of the nerve-fibres.

In summing up the above histological changes, it may be said that they consist in atrophy of the nerve-elements, and hypertrophy and hyperplasia of the connective tissue.

Special attention has been drawn of late to the various changes which indicate a choking of the lymph-channels of the cortex, whereby lymph-stasis is induced. These are—hyperæmia (dilatation) of blood-vessels, with increase of their nuclei, thickening of their walls, and accumulation of protoplasmic heaps in the subadventitial and perivascular spaces; the overgrowth of spider- (secondary glia-) cells, with distortion of vessel-sheaths produced thereby; adhesions between pia and cortex, causing occlusion of parts of the epicerebral space, and of the communications therewith of the perivascular lymph-spaces.

Changes similar in character to those existing in the cortex are found in the area immediately beneath the cortex, in the medullary substance, the basal ganglia, corpora quadrigemina, cerebellum, pons, and medulla; but these are less marked, and have not claimed the attention which the condition of the cortex has naturally received.

Degenerative changes are present in the nuclei and roots of the cranial nerves (the vagi particularly, which helps to explain cardiac and pulmonary disorders), with evidence of vascular implication (pronounced hyperæmia, with thickening of the walls of the vessels).

The histological *changes in the spinal cord and peripheral nerves* are next to be considered. Changes occur in great variety in the cord, and are found in the majority of cases. Out of 145 cases Fürstner failed to find morbid alteration in 16 only. The tunics show chronic inflammatory changes; more especially there is evidence of chronic leptomeningitis; the pial septa are involved in this. In the cord itself there may be a diffuse degeneration, or irregularly occurring patches of degeneration (softening, sclerosis); more commonly the latter, in combination with changes in the columns. Degeneration is usually found in combination in the posterior and lateral columns, and most notably in the former; but sometimes singly, and then most frequently in the posterior columns. Fürstner finds that one side is regularly more affected than the other. The lateral columns are especially affected in the dorso-lumbar region; in the posterior columns degeneration is often seen along the whole length of the column, with degrees of local intensity. The anterior columns are but rarely affected, and never without disease in the posterior and lateral columns. The degeneration may be more intense in certain streaks; as in the posterior root-zones, or in Goll's tracts, or in the lateral pyramidal tracts. These degenerative changes consist in hyperæmia; the vessels being engorged, their walls thickened, and the nuclei of the latter increased. These vascular changes are especially prominent in the posterior columns. The connective tissue is increased, with nuclear proliferation, and enlargement and proliferation of the spider-cells; especially along the vessels. The medullated nerve-fibres show loss or swelling of myeline, the latter taking up the stain; or the myeline is granular, the axis-cylinders being interrupted or distorted. The cornua also exhibit changes in varying degree; there is atrophy and degeneration of nerve-cells, the latter being granular, swollen, translucent, with processes shortened (atrophied). The spinal nerve-roots (posterior and anterior) are also degenerate; their connective tissue is hypertrophied, from chronic inflammatory change; the nerve-fibres are atrophied. The lumb[illegible] sacral nerve-roots are believed to show the most marked change.

The peripheral nerves show, in varying degree, parenchymatous degeneration (degeneration of the medullary sheath; swelling and atrophy of the axis-cylinder) and atrophy; and overgrowth of the connective tissue. The degenerative changes are more marked in the nerves of the lower than of the upper extremities.

The ganglia of the sympathetic system have been found to exhibit changes indicative of degeneration of the nerve-cells and chronic inflammation of the connective tissue.

Degenerative changes are also found in the muscles, including the heart and diaphragm (Mott, Campbell). There is fatty degeneration with disappearance of the muscle-fibre and increase of connective tissue.

Conditions indicative of degeneration have been described (Klippel, Mickle, and others) in various viscera, as in the lung, stomach, intestine, liver, spleen, kidneys. Concerning the nature and origin of these changes (whether primary or secondary to nerve-degeneration) it is not possible at present to give precise information.

Pathogeny.—The disease is one involving the whole nervous system; though the cerebrum, and more particularly the cerebral cortex with the investing pia arachnoid, is in the vast majority of cases the earliest seat of disease, and the locality in which its effects are most pronounced. The morbid process is usually first manifest and most active in the frontal and parietal areas. In some cases the encephalon alone shows indications of disease. Occasionally, to judge by symptoms, the cord or bulb would appear to be attacked first, and the brain later. As regards structures outside the nervous system, it may well be that these are attacked independently of this system, and not merely involved secondarily. Thus the fatty changes which occur in various muscles cannot be summarily ascribed to degeneration in the nerves passing to them; on the contrary, examination of these nerves in some cases has failed to show changes which would account for degeneration in the muscles (Mott). It is not possible at present to affirm how far the various degenerative states of other tissues are secondary and due to nerve-degeneration (impaired nutrition), or to vaso-motor disturbance, dependent on degeneration of the central nerve-tissues, especially the cortex cerebri; or are primary and independent, ascribable—like the muscular degeneration—to the influence of a toxin.

The nature of the pathological process remains obscure. It has long been, and still is, a subject of debate whether the primary change be inflammatory, affecting the vessels and interstitial tissue—a meningo-encephalitis, or a parenchymatous degeneration—the specific tissue, the nerve-elements, being primarily attacked. According to the first view the degeneration and atrophy which the nerve-cells and fibres exhibit are due to pressure from increase in the surrounding connective tissue; the second view is that the inflammatory changes manifested by the supporting framework of the nerve-elements are merely secondary in nature, "the result of decay of the neurons, and the irritation of the products of their degeneration" (Mott). It will not be possible here to discuss these debatable propositions; it may be affirmed, however, that the view that the primary change occurs in the specific tissue, the nerve-elements, is at the present time receiving an increased measure of support. In favour of it is the fact that the frontal lobe—the highest level centre—is the part earliest affected, there being no apparent reason why, were the change primarily inflammatory, the temporal and occipital lobes should not be equally attacked; yet these parts exhibit lesions to a comparatively slight extent. Further, Zacher, investigating the brain in an early stage (fourth to eighth week) of the disease, found a high degree of atrophy of the nerve-fibres of the cortex, with very slight vascular lesion. Others (Colella, Awtowkratow) have described alterations in the processes of the cerebral cells, and in the cells of the nuclei in the bulb, where the blood-vessels have been simply dilated. It has to be remarked, however, that, whereas vascular and nerve lesions are commonly mixed without a clear preponderance of one morbid condition over the other, in some cases the former, in others the latter are the more evident; in other words, the process would appear to be sometimes mainly inflammatory, sometimes mainly degenerative.

At the present time attention is being directed to the inquiry whether certain of the lesions of general paralysis can be referred to the action of toxic substances. Mott has suggested that the lesions found in various muscles are ascribable to toxic influence; and the latter may also be responsible for morbid states of the cortex cerebri which underlie certain clinical manifestations, especially the convulsive seizures. Varieties of histological lesion and of clinical state would be ascribable to variations in the nature and method of operation of the poison, and in vulnerability of tissue in individual cases. Such toxin may enter from without, or may be elaborated within the system (auto-intoxication).

These considerations would throw light more especially upon the origin of certain complications of the disease. But in the cases of general paralysis in which a history of syphilis has been clearly made out, it is legitimate to suppose the introduction of a toxin which has impaired the vitality of the nerve-elements, and which is fundamentally responsible for the disease itself, by producing a vulnerability of nerve-tissue which allows the various determining factors to become operative.

The interference with the lymph flow, which occurs in the course of the morbid process, and which results from occlusion of the lymph-channels, as indicated by the lesions above described, must have a deleterious effect upon the nerve-elements, and hasten their degeneration. The morbid development of the spider-cell—one of the conditions instrumental in bringing about this occlusion—is a feature to which much importance has been attached by Bevan Lewis, by whom this cell is regarded as the distal extension of the lymphatic system. According to this writer, the spider-cells in general paralysis, and in other diseased conditions of the nervous system, act as phagocytes or "scavengers," multiplying upon and removing the degenerate nerve-elements; they are also destructive of the living tissue. The nerve-elements are replaced by fibrillar connective tissue. This replacement is sufficiently shown in sections. It is ordinarily held that the nerve-elements are destroyed by the compressing action of the newly formed sclerous tissue. But in the hypothesis to which reference has just been made the replacement is regarded as a genuine degradation, the sclerous tissue being formed out of the effete material afforded by the degenerate nerve-tissue.

Further research is, however, needed ere a critical opinion can be expressed upon these views.

EDWIN GOODALL.

Diagnosis.—It is not always possible to distinguish between general paralysis and some other forms of mental disorder; each stage of the disorder has its difficulties. No single symptom can be considered as pathognomonic; to establish the existence of the disease we must prove the presence of both bodily and mental symptoms, which on the whole are progressive. In forming a judgment it is well to remember that there is no form of insanity which may not be associated with general paralysis. In examining male patients of middle age the question of general paralysis must always be taken into consideration. General paralysis is rarely detected in its earliest stages, which stages are not recognised until the disease is fully established. Neurasthenia may be the starting-point of general paralysis; therefore in every case of extreme nervous weakness it is well to examine the state of the pupils and the knee-jerks, and to look for any evidence of the emotional instability and change in temper which are common in this disease. If hysteria occur in middle-aged men it is well to look out for the above signs of degeneraion; and in both neurasthenia and hysteria changes in articulation and n writing must be closely observed.

Convulsive or paralytic seizures of a transient kind must not be overooked, as they often precede the more marked signs of general paralysis. The occurrence of such fits or palsies for the first time in men without ardiac or renal disorder, the rapid passing off of the symptoms, and the presence of some such physical signs as pupillary inequality, or sluggishness with defects of expression, suffice to clear up any doubt between epilepsy and general paralysis. The perpetration of criminal acts by men previously moral is often found associated with early general paralysis; but in a court of law some marked symptom such as fits, pupillary changes, or well-marked change in character of a progressive type not attributable to alcoholic excess, must be relied upon. If simple depression or slight hypochondriasis suggest early general paralysis, physical signs of degeneration must be looked for; and I think the most important thing is to observe whether there is any inconsistency between the mental complaints and the physical states; thus the hypochondriacal general paralytic complains, but still gains flesh and appears well.

The diagnosis in the acute stage rests chiefly between mania of various kinds and the maniacal onset of general paralysis. I do not think it is possible to make a distinction between acute delirious mania which may end in general paralysis and acute delirious mania which may pass off, or may end in death.

In all cases of delirious mania not due to alcohol, in which there is a history of syphilis, it is well to be on the look-out for general paralysis. I think in the latter there will probably be found more marked alteration in the articulation, and more marked tremor about the muscles of expression; but in acute delirium also these may be present to some extent. Pupillary inequality may aid diagnosis, but in either case the pupils may be dilated and rather immobile. In ordinary acute mania there may be great difficulty and delay in coming to a definite decision; and the difficulty is the greater when the mania depends on alcohol. As a rule in mania the sleeplessness is better marked, and there is rather more persistence in one line of thought than in the acutely maniacal stage of the general paralytic, in whom there is greater mobility and more rapid change of ideas. In general paralysis, again, the exaltation is more marked and more variable than is common in mania. In general paralysis drugs and alcohol seem to have a greater effect than in maniacal cases. If in mania we meet with great variability, intolerance of drugs, pupillary inequality, and change in speech and expression, general paralysis may be suspected. If the earlier symptoms be melancholic there is nothing characteristic in these alone. The melancholia may be of any form; but if with melancholic symptoms there is "physical inconsistency," that is, if the patient gain flesh, though still complaining that he is ruined, or is a miserable sinner, and if it be found that his pupils are unequal and indolent, and his speech defective, then general paralysis is to be feared. The occurrence of convulsive seizures often clears up the diagnosis. In some cases when stupor occurs early in general paralysis great doubt may arise: in these cases, if the patient gain in flesh without losing the apparent melancholy, we should search for inequality of pupils and for tremor about the facial muscles; but, again, it is common for the nature of the disease to remain unsuspected till convulsive seizures occur, or till the patient passes into a state of excitement. It is noteworthy that the majority of melancholic general paralytics have distinctly hypochondriacal ideas—the notion that their bowels are occluded being a common one.

In progressive dementia there may be great difficulty, for the mental symptoms are exactly the same in this and in general paralysis; but, as a rule, the progress is more rapid in the latter than in ordinary dementia: the latter, again, is more often associated with age or some definite cause of physical weakness, while general paralysis occurs in middle life. If in progressive dementia there are signs of muscular weakness—such as tremors and pupillary inequality and defective articulation, general paralysis must be suspected. The cases of simple general paralysis seen in general hospitals are usually of this type; convulsive seizures frequently reveal their true nature.

Alcoholic disorders and general paralysis.—As already pointed out, the symptoms of general paralysis are also the symptoms of alcoholism; hence, unless the history be very clear, it is impossible to form a correct opinion at once. In some cases, indeed, intemperance and increased nervous instability are symptoms of general paralysis. The excitement of delirium tremens resembles the delirious onset of general paralysis; and it is well to remember that delirium occurring in a man as the result of comparatively slight alcoholic excess may depend on this kind of degeneration. The acute alcoholic patient, as a rule, sleeps worse than the general paralytic, and has much more marked visual hallucinations of the terrifying kind. The diagnosis must be made from the history, and the existence of some change in the pupils, speech, or writing; but in spite of the most careful consideration mistakes will occur, and time alone can determine the nature of the disease. I think that in every acute case of mental disorder in which there is any suspicion of alcoholic causation time for observation must be insisted on. Convulsive seizures may result from alcoholic excess, and I do not think anything but time can decide on the cause of these fits. Chronic alcoholism resembles the second stage of general paralysis in many ways; but as a rule there is more evident loss of recent memory in the alcoholic than in the general paralytic; and the drinker "makes believe" more, talking of what he has been doing and seeing, when he has in fact never left his room. There is no pupillary change, the reflexes may be normal, and, though the facial expression is changed, there are not the tremor and the defective articulation which are so common in general paralysis.

Lead-poisoning will produce any of the ordinary symptoms of general paralysis, and may, indeed, be the cause of the disease; we must make sure, therefore, from the history and the physical signs that lead is the cause. The blue line on the gums, the tenderness, and the early paresis of the extensor muscles of the arms are the chief points of distinction. I have never seen a case in which morphia or any other vegetable alkaloid has produced symptoms which could be confused with general paralysis; but I have seen several chronic takers of chloral, with loss of mental and muscular power, who were not easily distinguished at first from general paralytics. In these, as in drunkards, removal of the drug and careful observation are our only safeguards. Influenza has in many cases set up degenerative changes which turned out to be general paralysis; it is therefore not to be forgotten that general paralysis may follow rapidly on influenza.

The *convulsions of kidney disease* may be mistaken for general paralysis; and, in some cases of alcoholic kidney disease, dementia may have been slowly coming on before the fits, and difficulty may thus arise; but, as I have said, albuminuria is very rarely met with in general paralysis.

Syphilis and General paralysis.—As I have already said, syphilis is a common cause of general paralysis, and the affinities between some cases of brain syphilis and general paralysis are very well known. The chief point is this, that in syphilitic brain disease apart from general paralysis we expect to do good by specific treatment, whereas in general paralysis this treatment fails; the nocturnal headache and oculo-motor palsies are very common in brain syphilis, and very rare in general paralysis. In cases of progressive mental decay, in which there has been syphilis with symptoms pointing to coarse intracranial lesion, the implication of any cranial nerve is against general paralysis; though it must be remembered that in general paralysis such a nerve implication may occur, especially in the prodromal stages. That a widespread syphilitic cerebral arteritis is often mistaken for general paralysis of the insane has been pointed out by various observers. Locomotor ataxy may be due directly to syphilis or be a part of the general paralysis.

I do not think it necessary to describe the so-called *pseudo-general paralysis*. Suffice it to say that in many cases of syphilis there are brain symptoms which prove not to be progressive, and which are not part of a steady process of decay.

Tumours of the brain, whether cancerous, syphilitic, or gliomatous, may lead to difficulties in diagnosis. A history of cancerous growth elsewhere might assist the diagnosis; but the presence of local headache, of vomiting, of optic neuritis are more important distinctions. The better-marked effects left after convulsions due to brain tumour are likewise important. The mental state of the patient with brain tumour is, as a rule, one of apathy rather than of excitement or disorder.

Tumours about the pons or cerebellum are also at times difficult to distinguish from general paralysis; but, as a rule, the staggering gait is greater than with general paralysis of the same duration; moreover optic neuritis is pretty sure to be present, with vomiting and general affection of certain groups of neck muscles.

Disseminated sclerosis may in some cases lead to confusion; when this disease was first demonstrated in England, by Dr. Moxon, I saw many cases of general paralysis, in general hospitals, which were regarded as insular sclerosis. Insular sclerosis usually occurs in young persons, and its progress is slower; there is a difference in the speech which is more staccato, the movements are rather jerky than tremulous, at least for a time; there is often nystagmus, which is very rare in general paralysis, and muscles are often picked out in various groups; the special degradation does not follow the lines of latest and most special development.

Paralysis agitans may be associated with mental weakness, but, generally speaking, the character of the movement is quite unlike that met with in general paralysis; the age of the patient also aids the diagnosis.

Sunstroke may give rise to nearly all the symptoms of general paralysis; and a history of sunstroke occurring in a tropical climate and apart from alcoholic excess must make one hesitate in giving a final judgment on a case.

Epilepsy is hard to distinguish from general paralysis. If the fits alone are considered, there is nothing distinctive about them; they may affect the same centres in the same way: but whereas epileptic fits usually pass in a short time leaving no paralysis, in general paralysis the parts affected are almost always paralysed or enfeebled for some hours or even for days after a fit. The epileptic fit, if the first, does not often leave much mental deterioration, while in general paralysis a fit often leaves the patient docile and quiet, however excited and grandiose he was before it. In some cases slight attacks, as of petit mal, occur in general paralysis; and in such cases the difficulty is the greater because there is but little mental change after or with them; in such cases you must depend on the pupillary or other general symptoms of general paralysis.

Apoplectic fits occur in general paralysis, and, as with epilepsy, they cannot at first be distinguished from true apoplexy; as a rule, however, they pass off in the course of a day or two—thus proving that, though implicating one-half of the brain and body, the lesion was rather functional than organic. The frequency of fits of this kind may also indicate their nature. I have known a patient have a dozen severe apoplectic fits, yet recover a fair amount of mental and physical power. In some cases apoplexy proper may appear to be the starting-point of the general paralysis.

Probably the greatest difficulty arises in the cases of men who have many of the symptoms, both bodily and mental, of general paralysis, but who prove later to be suffering from mental and moral perversion, and not from progressive disease. I can best make this clear by describing a case.

A middle-aged man of very neurotic family, married, with no family, with no history of syphilis, who had led a moral, respectable life, but who had suffered from various gouty symptoms, had a severe bodily illness from which he slowly recovered. After this he was a changed man, his conversation was no longer refined, he spoke openly of sexual matters, and made ribald jests before his wife and family. He then got entangled with a notoriously profligate woman, and was seen with her openly. He even proposed to introduce her to his wife and friends. He could not see the impropriety of his conduct, but suggested that his wife should follow similar ways. He retained his affection for wife and family, although still leading this most immoral life.

This state of things went on for many years, the man saying that now at last he had learnt how to enjoy life. In such a case I have seen pupillary inequality, unilateral sweating and some hesitation in speech; but no defect in gait or expression, no loss of memory, and no progressive decay.

I can only repeat that although such cases are almost always considered to be general paralysis, yet the patients may go on unchanged for many years. I have heard them described as cases of satyriasis.

Criminality or General paralysis.—First, as an early symptom of the disease, the patient may lose control and be guilty of impulsive criminal acts; in a moment he may commit a murder or cause grievous bodily harm; he may destroy property or do other acts which are simply due to his defective power of control; like many persons who have suffered from injury to the head, he may be more easily affected by alcoholic and similar stimulants.

Suicide may result from similar want of control.

Wrongful desire of one kind and another may arise, to be gratified without regard to social law; thus the patient may steal, though in my experience the general paralytic who commits theft does so from a conviction of his great wealth, and his right to all he may desire.

To complete this part of my subject I will now repeat one or two points already referred to.

Male hysteria may simulate general paralysis, yet general paralysis may begin as hysteria. Exaltation of ideas may occur in mania, especially if due to alcoholic exaltation; it may be the natural result of defective control in youth—an overgrowth of conceit, or it may result from delusions with an apprehension of being noticed or watched; it may be a steady growth of the imagination, the "may be" becoming a reality; but in general paralysis, as a rule, the exaltation is general and of a benevolent character.

It is difficult to tell whether a patient be suffering from mania of the double form, or from general paralysis of the double form; and time alone can settle the question.

Paraplegia in its various forms may be associated with general paralysis, and set in before, during, or after the mental symptoms; therefore in every such case the mental as well as the bodily weakness must be studied.

Sexual faults or crimes are commonly met with; these acts may arise from exaggerated or inflamed desire, or from loss of control. Indecent exposure may arise from unnatural desire or from simple forgetfulness. Rape and assault on children, indecent and criminal assaults, are apt to occur likewise. In the earlier stages of the disease there is almost always great increase of sexual desire, and some increase of sexual power may be present; and this may last for a considerable time.

The period when criminal acts are most likely to be done is before the full development of the more marked symptoms, or in the first period of their manifestation; hence it is of great importance that careful examination should be made for physical signs of degeneration in suspected individuals.

As far as *mental capacity* is concerned, it is certain that some general paralytics become unfit to dispose of their property long before they can be certified as insane; for in the earlier stages of the disease great alteration of temperament and disposition takes place as well as a tendency to act without due judgment; and later the memory may be defective also. Thus, as the result of progressive brain disease, a patient may be alienated from his relations, attracted by some designing person, probably a woman, and, as the result of these perverted feelings, may make an unjust will. On the other hand, a general paralytic can make a perfectly reasonable will, even in the late stages of the disease, provided the symptoms chiefly affect the muscular side of him. It was even allowed in the case of Crabtree, tried in 1894, that a general paralytic during a period of remission may make a valid will, though he had been previously in a state of apparent dementia, and had had fits.

A general paralytic, indeed, may be guilty of crimes of various kinds as a result of his disease, but may be held capable, nevertheless, of making a will even at an advanced stage of the disease (see Brown *v.* Penn, Nov. 1895).

Prognosis.—This is rather concerned with the probable course and duration of the case than with the result; I can claim very few recoveries from undoubted general paralysis. As I have said, sometimes early threatenings of the disease, occurring in likely subjects, may pass off. In some cases remissions may be so complete for many years that recovery is alleged; but the end of nearly all these cases proves that the respite was but arrest or remission. I will not say that no case of general paralysis ever ends in recovery, but it is very rare. The more acute the onset, the greater the prospect of remission; and the stronger the neurotic heredity, the greater, I believe, is the prospect of remission, and of the disease running an irregular course. It is very rare for more than one complete remission to occur in any case, save in those of the double form. In a few cases the quiet demented stage may last for years. General paralytics live longer in asylums than in their homes; they live longer in small asylums than in larger ones, and they live longer in the country than in towns. It is not possible to say whether a particular case will run a short course or a long one; yet as a rule the higher the temperature the greater the danger of rapid decline. The presence of fits generally hastens the progress; but in rare cases fits seem to arrest the disease for a time, or even to lead to remissions. Though a patient may have been quiet and placid in the earlier stages it is not certain that he will not have a maniacal outbreak later. The cases of general paralysis of the double form, or of the circular form, generally last longer than others, their duration being extended by the restful periods and prolonged remissions. A clear history of syphilitic infection within five years will give more prospect of amelioration; but, however distinct the history of syphilis, if of many years' standing I do not think it really affects the prognosis; that is to say, if the diagnosis of general paralysis be confident.

Treatment may be divided into general and special. The first consideration is whether the patient can be properly treated at his own home, or at any rate out of an asylum. Seeing that these cases are almost hopeless, it is well in all cases to decide at once in favour of removal from home to definite asylum care, except in those of simple progressive dementia without excitement, very distinct depression, or marked delusion. The treatment of a very wealthy general paralytic may be undertaken in a private house with large grounds away from a city; but even then, in my belief, it is neither the best nor the kindest treatment. Whether away from home or not, the patient's nearest relations, more particularly his wife, should not live with him. The less emotion there is in the life of the general paralytic the longer will his life last. Indulgence of the sexual passion is injurious, and the prospect of children begotten by such a father is gloomy. The wife in such cases, if installed as nurse, will probably break down in health; and I frequently see neurasthenia as the result of wifely devotion to general paralytic husbands. Sea voyages, though so often recommended,—only it seems to me in order to shirk the alternative of an asylum,—are rarely, I think, of any service.

The golden rule of treatment is complete rest. The simplest country life, with as much sleep as the patient can take, is to be desired; only moderate exercise must be allowed, and of course riding and cycling have to be given up. During periods of remission it is difficult to prevent the return of the patient to his own home; but this return is almost always followed by relapse. It is better to send the patient away for the interval; possibly for a short sea voyage, or else to the house of some medical man living in the inland country. The diet of the general paralytic must vary with the stage of the disease; in all I think alcohol is to be avoided. The simpler the food the better; fish, fruit, and plain food is better than much meat and fancy dishes. If any stimulant be found necessary it should be as weak spirit and water or light wine. Tobacco may be of service in the earlier stages of restless excitement; but there is a danger that the patient may smoke too much.

The general paralytic must be warmly clad, and his clothes carefully looked after, as he is very careless of himself.

I do not think that electricity in any form is of service. Specific medical treatment has made but little progress as yet. If antisyphilitic treatment has not been fully tried, I recommend, if the patient be quiet enough, the course of treatment at Aix-la-Chapelle. If this has already been tried, we know of no specific. In some cases counter-irritation along the spinal column has been tried, and said to be of use. Iodine liniment to scalp and spine has been recommended. Surgical treatment has been tried with the idea of relieving pressure, but the results of trephining have not been encouraging. I trust that sooner or later some antitoxin will be found which will aid in defeating this malign disease. Calabar bean was used for some time, but I cannot report favourably on its action; nux vomica is useful, with or without quinine and hydrobromic acid; I cannot say that setons in the neck have produced any good effect. In ordering drugs for general paralytics we have to remember that they are very readily affected by strong remedies; I have known minute doses of alkaloids produce in them very alarming symptoms. If there is much sleeplessness I prefer paraldehyde in drachm doses to any other drug; I do not like morphia or hyoscine; trional or sulphonal generally fails to produce sleep. If the patient is in fits we may use chloroform or nitrite of amyl; and nitro-glycerine, too, has been found of service. If the *status epilepticus* continue to an alarming extent, I advise an enema containing 20 grains of chloral with 20 grains of a bromide. It is necessary in these cases to consider details very carefully; thus it may be necessary to empty the bladder and perhaps to wash it out. Soreness of the skin may be prevented by great attention to keeping it dry, and also by the application, night and morning, of hot water and dry Castile soap.

f hæmatoma occurs it is best treated by immediate and vigorous listering; this failing, it may be necessary to slit the part open and emove the clot.

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INFANTILE CEREBRAL DEGENERATION

Syn.:—*Arrested Cerebral Development—Amaurotic Family Idiocy. A Family Form of Idiocy. Agenesis Corticalis.*

Definition.—A rare and fatal disease of infancy characterised by progressive mental and physical enfeeblement, associated with symmetrical changes at the macula lutea which are pathognomonic.

History.—Attention was first called to this disease by Mr. Waren Tay in 1881; and since then twenty-eight cases have been published, including Mr. Tay's patients. Ophthalmologists have contributed most of the cases on record, as the peculiar and characteristic ocular changes form a prominent part of the clinical picture of the disease. B. Sachs of New York, Peterson, Hirsch, and one of us (Kingdon) in this country have alone been successful in obtaining autopsies; the latter observer being particularly fortunate in that he has been able to supplement his clinical observations by autopsies and subsequent microscopical examination of the nervous system in three members of a family of seven, five of whom were affected.

Causes.—A curious circumstance, which appears to be of etiological significance, is that, of the published cases in which the nationality is stated, the patients have all been Hebrews. In addition to this racial peculiarity, there is distinct evidence that the condition belongs to the category of family diseases; more than one child in the same family is, as a rule, attacked, though others may remain perfectly healthy. Of the twenty-eight cases recorded, ten were in boys, and fourteen in girls; in four cases no mention is made of the sex.

Syphilis and other hereditary diatheses appear to play no part in its production; nor is there any evidence that consanguinity of marriage has any great influence in this connection, though it has been a possible factor in some cases. One or other parent has been neurotic in some instances, and injury during pregnancy has been noted, but this has been exceptional. No proximate cause after birth has yet been discovered, with the exception of a doubtful injury in a few cases.

Symptoms.—Children who afterwards show evidences of this disease are born at full term, are then, as far as can be judged, perfectly normal, and so remain until the end of the third (as a rule), or it may be as late as the sixth month; up to which time development progresses naturally. The first symptom to attract attention is an inability to sit up properly, and to keep the head from falling backwards; in addition to these evidences of weakness of the muscles of the neck and back, there is often a suspicion that vision is impaired; but usually no definite ophthalmoscopic changes can be made out so early.

As the disease progresses the child becomes apathetic, takes little notice of what is going on around it, and wears the vacant expression of mental enfeeblement. It becomes less and less able to sit up, and quite unable to hold up its head, which consequently falls backwards. It lies in bed as it is placed, and has no power of moving from one position to another. Objects placed in its hands are grasped feebly and soon dropped. The muscles become soft and flabby, but in the only instances in which they have been tested electrically in the early stage of the disease they responded to both the interrupted and constant currents. Soon there is evidence that the pyramidal system is implicated; slight extensor or flexor spasm appears in the limbs, and the tendon jerks become too active.

Later in the course of the illness the enfeebled muscles begin to waste; all the muscles of the body sharing in the atrophy, which eventually becomes extreme. To this muscular wasting, which is general and is confined to groups of muscles, there is added an increasing degree of spasmodic rigidity which causes retraction of the head, and distortion of the different segments of the limbs,—resulting, for instance, in inward rotation of the arm, pronation of the forearm, flexion of the thigh on the abdomen with marked abduction of the limbs, over-extension with partial luxation at the knee, extension of the foot, and so on. As the contracture increases the child evidently suffers much pain, which is increased by the spasmodic jerking of the limbs which occur from time to time. Death may result, however, before spasmodic contracture has taken place; or rigidity may have been occasional, no spasm being permanently present.

Convulsions are so rare that they form no part of the usual clinical picture of the affection, but have been observed as terminal manifestations of the illness. Cutaneous sensibility and the superficial reflexes are preserved, and the former has occasionally been found increased, so that the child starts at the slightest touch. The special senses, with the exception of vision and hearing, remain unimpaired throughout the illness. Sight fails gradually until total amaurosis results from optic atrophy, which has all the characters of being primary, and in the majority of cases is undoubtedly so; though Mr. Waren Tay has observed slight neuritis in the early stage of the optic nerve affection. In addition to this, ophthalmoscopic examination reveals a peculiar and distinctive appearance in the region of each yellow spot. At first a suspicious-looking haze is seen in the macular region, but subsequently a whitish gray oval patch is seen, almost twice the size of the optic disc, with softened edges, and with its long axis lying horizontally, very slightly raised above the general surface of the retina. The fovea centralis is seen as a dark cherry-red spot in the centre of this patch. Once established, these appearances remain unaltered throughout the whole course of the affection, and were present in Koller's patient, aged four years.

There is no evidence that either the cranial nerves—other than the second pair—or their nuclei are affected, speaking generally. The pupils are equal, and, as optic atrophy becomes advanced, they are dilated and inactive to light.

Strabismus has been noted exceptionally, as has nystagmus also; but both phenomena must be rare: towards the final stages of the illness, however, slow rhythmical movements of the eyes from side to side may occur. The only other points calling for comment, as far as the cranial nerves are concerned, are that hearing sometimes appears to be abnormally acute, a phenomenon too frequently observed to be merely accidental; and there may be difficulty in swallowing for some time prior to the fatal termination of the case.

Apart from any accidental complications, the thoracic and abdominal viscera are, as a rule, normal; the appetite good, the bowels regular, the urine natural, and the pulse, respirations and temperature normal. In Sachs' first case, however, the gastric mucosa early refused to perform its functions properly; and some elevation of temperature has been noted in the terminal stages of a few cases.

The disease has ended fatally in most of the recorded cases, and in those in which this result had not been reached, the condition of the patients at the time of publication made it evident that there was not likely to be any departure from the general rule; the only known exceptions being Koller's case of a girl aged four years, and a child aged four and a half years cited by Peterson. The duration of life varies from one and a half to two and a half years, is usually less than two years, and may (quite exceptionally) be prolonged beyond this, as in the two cases already referred to. The final stage of the affection sometimes resembles the same stage in cases of general paralysis of the insane. Death may occur before much emaciation, or evidence of spasmodic rigidity of the limbs. But usually the child becomes pale and shrunken, more and more lethargic and exhausted until marasmus becomes extreme, consciousness is lost, and death closes the scene. In exceptional cases, instead of this more usual form of death from marasmus and gradual exhaustion, the termination may be unexpectedly sudden.

Morbid anatomy.—The cerebral and spinal meninges usually present no abnormal appearances; though in Sachs' first case a few slight adhesions of the former were noted. The convolutions and sulci of the brain are normally arranged; but in the case just referred to there was some abnormal fissuration which was regarded as indicative of a brain of low development, an abnormality present in Peterson's case also. The sulci are wide in some cases, pointing to a certain amount of atrophy of the convolutions; but this atrophy varies in different cases. The ventricles are normal, and there is no excess of cerebro-spinal fluid in them; though there is some compensatory œdema of the meninges. The essential change met with on microscopical examination is degeneration of the neurons of the cerebral cortex, more especially those of the central convolutions. The pyramidal cells are seen in all stages of degeneration. Nissl's method of staining reveals various degrees of alteration in the size and shape of the cells; some are so distended as to become balloon-shaped, others preserve some traces of their original shape, while in some the changes are sufficiently advanced to lead to disintegration and vacuolation of the cell. The stichochrome granules disappear from the cells, and while, in some instances, fine chromatic granules are seen scattered through them, in others they are entirely without chromatophile substance. Phagocytes in the vicinity of the degenerating cells, and around the vessels, contain in their interior a large amount of the chromatophile substance which they have derived from the nerve-cells.

The Golgi-Cajal method of preparing portions of the cortex for examination also reveals clearly that degeneration of the neurons is in progress; in some cases the cells are variously altered beyond recognition, and all stages of degeneration are seen in the axons and dendrons. In most cases these processes are broken off from the cells, while, if still continuous with them, the characteristic beaded appearance of degeneration is seen along the course of the axis-cylinder. Evidences of similar changes are met with in other parts of the cortex, but they are not nearly so marked as in the region of the central convolutions.

By the Marchi method of preparation intense degeneration of the neurons may be traced through the corona radiata and internal capsule to the pyramids; this, and other methods of staining, reveal pronounced degeneration of the direct and crossed pyramidal tracts in the spinal cord on both sides. The perivascular lymphatics of the vessels in the corona radiata are full of fatty debris stained black by the osmic acid in Marchi's fluid. There is no evident change in the neuroglia; no sclerosis, no alteration in the walls of the blood-vessels, and no infiltration of the tissues in the neighbourhood of vessels. Degeneration of the cells of the basal ganglia, nuclei of cranial nerves, olivary bodies, and the anterior and posterior horns of the spinal cord have been found by Hirsch; the cells of the cerebellum alone showing but little change. Some degeneration has been found in the lemniscus on both sides, the descending roots of the fifth nerve and superior cerebellar peduncles. No changes have been found in the peripheral nerves.

The ocular changes consist in atrophy of the optic nerves, without any evidence of antecedent inflammatory mischief. Holden has found degeneration of the ganglion cells of the retina, otherwise nothing had been found to account for the changes seen in the macular region other than a thickening of the retina at this part, a change due to a spacing out of the outer molecular layer, as if by œdema (Treacher Collins).

Pathology.—While it is possible that the disease may be congenital in origin, neither in the clinical history nor in the morbid appearances met with after death have we any evidence of this. Nor do we find evidence that the affection is one of arrested development, as has been advocated by Sachs; for, while it is true that the changes met with lead to arrest of development and ultimate dissolution, it cannot be said that these changes in the central nervous system are themselves simply the result of arrest of development. On the contrary there is every reason to regard the changes as the result of a progressive degeneration of the neurons, such as might well result from the action of some toxin. The altered shapes of cells, regarded by Sachs as evidence of congenital mal-development, appear, as now investigated by more modern methods, rather to be the results of degeneration in normally developed structures; in none of the cases that we have examined has there been abnormal fissuration or other evidence of low cerebral development.

The advanced state of degeneration of the pyramidal tracts suggests the possibility that the changes in them precede those which are met with in the cerebral cortex; or that the changes in the cortex begin at a stage prior to the myelination of the pyramidal fibres. That the latter cannot be altogether the case is proved by the presence of many normally myelinated fibres in the pyramids, and of others in a state of recent degeneration with disintegration of their myelin sheaths. Again, that the former possibility is improbable is made evident by the fact that there is nothing in the clinical history of these cases to suggest that sclerosis of the pyramidal tracts precedes the affection of the cerebral cortex. There is no reason, therefore, why we should regard the morbid process otherwise than as one affecting the whole motor neuron from its beginning in the cerebral cortex to its end in the spinal cord; and, as the nutrition of the neuron depends upon the integrity of that part of it known as the cell, it is possible that the destructive process begins in the cell, and that the axon and dendrons suffer secondarily. On the other hand, the baneful influence may primarily attack the axon and dendrons; or all parts of the neuron may be affected simultaneously. Whether these changes depend on a deficiency or alteration in quality of some internal secretion, on the presence of some toxin, or on some other cause, remains for future research to determine. There is nothing in the histological changes to suggest an inflammatory process, syphilitic or other.

No satisfactory explanation of the relationship between the changes at the macula lutea and those met with in the central nervous system can be offered, unless it be that the ganglion cells of the retina are similarly affected to those of the cortex cerebri, and that they are thus affected has been shown by Holden. The occurrence of optic atrophy, on the other hand, is commonly met with in degenerative conditions of the central nervous system, though the mechanism of its production is not easy to explain.

Diagnosis.—When the changes in the region of the macula lutea are fully displayed they are so characteristic that any uncertainty as to the nature of the case can no longer exist. We know of no other condition in which progressive mental and physical enfeeblement are associated with these peculiar and pathognomonic changes. Even before the appearance of the macular changes the diagnosis is not difficult, though the affection may be then confounded with other infantile cerebral palsies. If a trustworthy history can be obtained, the pre-natal and natal affections are readily distinguished from this disease; the earliest evidences of which become manifest at the third month. It may be confounded with inherited syphilis; and the history of syphilis in a parent, and evidences of this disease, ocular or other, in the child must be sought for in attempting to arrive at a diagnosis. Ordinary congenital idiocy is distinguished by the absence of ocular symptoms and fundal changes, and such patients live for many years.

As regards its distinction from other post-natal cerebral palsies, and especially the family forms of these, reliance must be placed on the absence of convulsions; on the exceedingly gradual and general invasions of the paresis, which does not assume the form of a hemiplegia or a paraplegia; and, above all, on the presence of blindness, with the characteristic fundal changes. The possibility of confounding this disease with acute anterior poliomyelitis is too remote to deserve much attention; but should the question arise before the development of the changes in the eye grounds, the absence of altered electrical reaction of the muscles would exclude the spinal affection.

Prognosis.—With the exception of one case, recorded by Koller, in which the retinal changes were detected in an idiot aged four years, and the case referred to by Peterson, all the cases have ended fatally, and this usually within two years. Possibly as the condition becomes more widely recognised it may be found that more of the inmates of idiot asylums have been affected by this condition in infancy, and have escaped what appears to be the usual fate; but in the present state of our knowledge none but a gloomy prognosis is warranted. In the vast majority of cases as soon as the diagnosis is certain the prognosis is equally so. Moreover, the occurrence of a case of the kind in a family renders it highly probable that any children born subsequently may become similarly affected; fortunately this is not invariably the case.

Treatment.—No remedy appears to have any influence on the course of the disease, which is steadily progressive in spite of all the measures that have been tried for its relief. Mercury and iodide of potassium have both failed to influence its course; whether arsenic and similar remedies may prove more successful remains to be seen. If future researches should reveal any defect in connection with any of the ductless glands in these cases, the administration, in some form, of the particular gland affected might prove of service. So far the only measures of this kind that have been tried are the administration of pituitary gland and of cerebrine by one of us (Kingdon); both, however, with negative results. The same observer has made unsuccessful attempts to prevent the occurrence of the affection by administering iodide of potassium to the mother while pregnant. This was tried in two pregnancies; one child escaped, the other was attacked by the disease. We suggest that, in families where any case of this kind has been known to occur, the children should be weaned from the time of birth; on the chance that the mother herself, however healthy in appearance, may nevertheless communicate in her some deleterious product, chemical or other, which is capable of generating the disease in the suckling.

The most careful nursing, feeding and hygiene of such children are of course essential.

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THE CEREBRAL PALSIES OF CHILDREN

THE cerebral palsies of children are divided into two great classes—those in which there is evidence of the presence of paralysis when the child is born, and those in which, after a period, more or less prolonged, of apparently normal health and unimpaired activity, some form of paralysis appears. In the latter class the paralysis is, as a rule, hemiplegic in form; in the former both sides usually suffer: sometimes the legs only are affected, at any rate in a marked degree; sometimes both arms and both legs suffer; and in other cases one side only appears to be weak. Cases in which paralysis is present at birth are further subdivided into those in which the paralysis is the result of some difficulty or abnormality in the labour, and those in which it results from some condition arising during intra-uterine life; either primary disease or maldevelopment in the fœtus itself, or some morbid condition secondary to disease in the mother, or to injuries which she may have sustained during her pregnancy. By far the largest number are the result of abnormalities in the labour, the abnormality consisting usually in the length and difficulty of the labour, due to deformity in the mother or to malposition of the child. Nevertheless an unusually quick or precipitate labour may likewise give rise to birth palsy.

BIRTH PALSY.—**Symptoms.**—In this form of paralysis there is a variable distribution of weakness. Sometimes the lower limbs are affected alone, equally or unequally; producing the condition sometimes spoken of as "cerebral spastic paraplegia" or "congenital spastic paraplegia" ("Little's disease"). Sometimes the paralysis is of one side, affecting both arm and leg; sometimes both arms and both legs are affected; sometimes both legs and one arm only, or at least only one in a very obvious degree. In the limbs the weakness is usually associated with much stiffness. The gait, if the patient is able to walk, is characteristically spastic, each leg being dragged forward as a whole, and with considerable difficulty; if the upper limbs are affected there is, besides the weakness of the arms and hands, a great awkwardness in using them, probably occasioned by the spasticity. The knee-jerks are much exaggerated, but ankle clonus is not so frequently present as might be expected; and in the case of patients suffering from this condition it is usually present only in those who are able to walk. In patients unable to walk, in spite of the fact that a high degree of spasticity may be present, it is usually impossible to evoke it. In some patients the peculiar mobile spasm known as athetosis is present; and in patients in whom all four limbs are affected, athetosis affecting both sides—double athetosis—sometimes occurs. In this form spasm there is usually a cycle of movements, one position slowly and gradually passing into another. Sometimes the hand only is affected, and in the great majority of cases it is most affected; but the spasm may affect all the parts of the upper limb, and may also affect the leg. It involves the face in a few cases, and the platysma muscle may markedly participate in the affection.

Besides athetosis, or mobile spasm, another form of involuntary movement is sometimes present; a form very closely resembling the tremor present in cases of insular sclerosis, in that it is only induced during voluntary movement. This tremor is well marked when an attempt is made to carry out a definite movement with the affected limb. In carrying the affected hand to the face, for example, at the beginning of the act the tremor is very slight; but as the hand approaches the face it becomes more and more distinct and even violent, so that it is impossible for the patient to keep the hand in a position of rest. This condition has been unfortunately named "*chorea spastica*"; just as athetosis has been as unfortunately named "*post-hemiplegic chorea.*" In neither form of tremor is there, as a rule, anything more than a distant resemblance to the movements of true chorea; although certain cases of so-called hereditary chorea have undoubtedly been cases of birth palsy with double athetosis. But both athetosis and "chorea spastica" are probably more common in cases of infantile hemiplegia than in cases of birth palsy.

Paralysis or paresis of some ocular movements is also not infrequently met with; and this occasions a squint of varying character and intensity, apparently not the result of an affection of one muscle in one eye, but of a slight affection of the different movements in each eye. The sphincters of the rectum and bladder are only affected to the extent of being less easily controlled than normal. In patients in whom there is much psychical change, the calls are unattended to, and the habits are consequently dirty. Even in these cases, however, this is only the rule in the early years of life. There is rarely any affection of swallowing, although slobbering is not uncommon.

The *mental condition* varies much in different patients. In those in whom all four limbs are affected, there is, as a rule, much mental change: if the lower limbs only are paralysed the mental condition is frequently good; and in some instances the children are precociously sharp. When the paralysis is hemiplegic in form the psychical condition varies, being very defective in some, scarcely abnormal in others. In some cases in which the paralysis is only slight, much change is present; a result probably due, as will be pointed out later, not to the nature but to the position of the lesion. In all the cases mental development is retarded, and, as a rule, does not attain a high level.

Speech also is interfered with, and sometimes seems to remain entirely in abeyance; in nearly all, its development is retarded, and generally it remains long imperfect. Anything like true aphasia is, of course, scarcely to be expected, for this can only arise from interference with the functions of a speech mechanism already developed, and in cases of birth palsy we have to do with centres whose development has been interfered with, or entirely arrested.

Recurring convulsions are not so common in cases of birth palsy as might be expected, possibly because of the completeness and limitation of the destructive process which has been present. But in many cases a history of a severe convulsion is to be obtained, or more often of a series of severe fits, occurring a few days after birth. Occasionally such convulsions recur at intervals during the first few years of life, and in a small number of cases this tendency persists during the whole of life.

Deformities are apt to occur in consequence of the interference with development, and the malpositions encouraged by the form of paralysis. The most common deformity is some form of talipes; and, in patients in whom one side is weaker than the other, lateral curvature is almost inevitable, unless extreme care be taken.

Causes.—In the great majority of the cases of birth palsy, as I have said, the lesion is determined by some fault in parturition. As a rule there is a history of a long and difficult labour, in many cases the result of an abnormal presentation of the fœtus; and frequently of a labour necessitating the use of instruments, or of turning. Often also the child is born cyanosed, and not breathing; so that artificial methods of promoting respiration have to be resorted to. Not uncommonly also the child is born before full term. But, as already pointed out in reference to these cases, any injury to the child occurring in a labour in which instruments have been used, or in which operative interference has been resorted to, is not to be ascribed to the instruments used or the operation performed, but rather to those abnormal conditions which rendered interference necessary. Besides the long and difficult labour, the quick, or, in technical language, the precipitate labour also seems to have an effect in the causation of the condition under consideration.

As abnormal labours are much more common in primiparæ than in multiparæ, it is accordingly to be expected that birth palsy will occur much more frequently in first-born children: this is actually the case in so large a majority of instances as to place the fact outside the region of mere coincidence. Two other facts come out in a series of cases of this character; namely, that of children affected with birth palsy there are twice as many males as females; and that the child of an elderly primipara is more liable to be afflicted than is the first child of a younger woman. These two facts lend support to the view which ascribes the great majority of cases of birth palsy to mechanical difficulties in the process of parturition; for in the former case the larger size of the child, in the latter the greater rigidity in the maternal parts will tend to increase the difficulties of the labour.

There is some evidence that inherited syphilis is sometimes a factor in the production of this condition. It is very uncommon to see a child the subject of birth palsy who has any of the recognised signs of congenital syphilis; but it is quite possible, of course, that a syphilitic child may suffer from birth palsy if it has been exposed also to the causes of this condition; and it is likewise probable that inherited syphilis, through affection of blood-vessels or membranes, may occasionally give rise to a morbid process which causes in the child symptoms similar to those which we have under consideration.

Morbid anatomy.—There is little doubt, from the cases which have been examined after death, that the condition upon which the paralysis depends is, in most cases, hæmorrhage occurring under the dura mater, and so affecting the cortical structures as to abolish or interfere with their function. From what has been said as to the causation, it will be seen that such a condition is what we should expect; and there seems no reason to doubt that its occurrence is due to the rupture of vessels in consequence of the mechanical difficulties in the act of parturition. The situation of this hæmorrhage gives the clue to the distribution of the paralysis. As a rule, as I have already said, the legs are more affected than the arms, sometimes they are affected alone. In such a case the hæmorrhage will be found on each side of the longitudinal fissure, spreading in each cortex over the area in which the leg movements are chiefly represented. If we suppose the hæmorrhage on one side to spread further, so as to destroy or impair the area in which the arm movements are represented, a condition would be produced in which both legs and one arm would be affected; if the hæmorrhage spread still further, one side of the face would be affected; and if it spread on each side, so as to affect both arm areas as well as the leg areas, a condition would be produced giving rise to paralysis of both arms and both legs. In cases in which the speech function is in abeyance, either completely or partially, the lesion is of the third frontal convolution on both sides or only on one; and it will readily be understood that the ocular movements may be interfered with if the lesion spread forward so as to involve the area related to these movements; thus a variable squint may be produced. I repeat that a varying degree of psychical defect is found to occur in children the subjects of birth palsy; and it may reasonably be assumed that this depends upon injury to that part of the brain which is probably most intimately associated with psychical processes—the præfrontal area.

This morbid condition is only distinctly recognised in cases in which death occurs in the early stage. Such, however, are not common; and when death takes place after the lapse of some years, the condition discovered varies considerably. Sometimes it is what is known as porencephalus, a condition in which there is much cystic formation surrounded by cerebral substance more or less altered: sometimes there seems to be a sclerotic condition of the brain itself, and in other cases a condition resembling pachymeningitis, due to the effused blood having become organised and altered. In short, from the condition found in such cases it would be impossible to discover the nature of the original lesion, were it not for the information gained from the examination of recent cases. It is also held by some writers that in certain cases, those especially in which the child is a premature one, the resulting condition is due to incomplete development of the pyramidal tracts. In such cases it is said that gradual improvement usually follows, and may go on to allow of an almost normal power of locomotion.

Diagnosis.—The diagnosis of the condition will not, as a rule, present many difficulties. The onset at birth, although it may not be noticed until a few days after it, and usually the history of some difficulty of abnormality at birth, or at any rate that the child was the first-born, are the points on which a diagnosis will depend. Infantile hemiplegia may be similar in the physical condition, or in the psychical symptoms; but in this disease the onset is later, it comes on in a previously healthy child, it is ushered in with a convulsion or convulsions, and it is strictly hemiplegic. The spastic paralysis due to caries may closely resemble cerebral spastic paralysis; but in the former condition there has been a period during which the limbs were used normally. Of infantile paralysis also—acute anterior poliomyelitis—the same may be said; and in this condition the muscular wasting, flaccidity, and loss of reflex action and of faradic irritability in the muscles will sufficiently distinguish it.

Prognosis.—Such a condition as that described entirely precludes any hope of complete recovery. The degree of recovery which takes place naturally depends on the extent and situation of the lesion; and will be greater when this is limited to the leg areas on each side of the longitudinal fissure. A patient with this condition will be late in acquiring the ability to walk, and even at the best will always walk stiffly and imperfectly; but the arms will be useful limbs, and the mental and intellectual powers will not necessarily be impaired at all, may even be rather above the average. In cases in which the upper limbs are affected there is, as will be evident from what has already been stated, a much greater likelihood of mental impairment accompanying the bodily ailment; and where such mental defect is present the improvement will not be great, at any rate in reference to that particular symptom.

But physical improvement takes place in all these cases. Often, although the children attain the age of five or six before they can walk, many of them, in whom the prospect of walking seemed out of the question in an early stage, do ultimately walk, imperfectly it is true. In short, physical improvement takes place in all, and the degree of this can only be estimated by ascertaining actually how much impairment is present; mental improvement also takes place even in children apparently quite imbecile at first. But in cases in which mental impairment is present, a normal mental condition must not be hoped for, any more than a perfect physical condition is to be expected in any case.

Treatment.—A child affected with birth palsy naturally requires very great care. Good and intelligent nursing is essential; suitable food and warm clothing are even of more importance than in the case of ordinary babies. No effort should be spared in attempting to develop the physical and mental capacity; for much may be done in this way to increase its powers. But such training is apt to be discouraging because of the slow, almost invisible progress which takes place, and it is well, therefore, that the nurse should not expect very rapid progress. Gymnastic exercises, such as will assist in increasing muscular power, rubbing of the limbs and passive exercises are of much importance. Cod-liver oil, cream, and fat in an easily digested form are to be regarded as articles of diet rather than as medicines.

Attention also will be given to measures calculated to prevent deformities, such as talipes or lateral curvature; or, if they have already occurred, to the operative or mechanical treatment which will tend to correct them, or at least to minimise their effect.

INFANTILE HEMIPLEGIA.—Infantile hemiplegia is the name applied to that paralysis of one side of the body which occurs in the first six years of life. Although in its general symptoms, except those occurring at the onset of the attack, it closely resembles the hemiplegia of adults, yet there are some distinct differences. The majority of these, however, are to be referred to the early age at which it occurs, and the consequent interference with development both of the brain and of the affected limbs. A distinction must be drawn not so much as regards symptomatology, but in reference to pathology, between cases of infantile hemiplegia proper, which is probably a peculiar disease, and cases of hemiplegia occurring in children in connection with acute disease; especially with rheumatism, scarlet fever, diphtheria, or typhoid fever.

The hemiplegia of infancy occurs during the first six years of life, but much more frequently in the first two years than later. Of forty-two cases, nineteen occurred in the first year, sixteen in the second, three in the third, three in the fourth, and only one in the fifth. The illness begins as a rule with general malaise and feverishness, and this culminates in a severe fit, or a series of convulsions. These are generally unilateral, but may spread so as to affect both sides. They may persist with but slight intermission for several days; and after the fits have ceased it is found that the child is paralysed on the side on which the convulsion was most severe, or to which it was limited. The resulting paralysis is permanent, although in degree it may be modified; and as a rule a considerable improvement from the condition immediately subsequent to the fit is to be looked for. If speech were present at the time of onset of the fits, it is usually interfered with at first, being either completely lost or rendered much less perfect than it had been before. Anything like the true aphasia of adult life is rarely if ever present; and it is doubtful whether the name aphasia is strictly applicable to the condition which results. Permanent speechlessness occurs in some cases, even in children who had already acquired the faculty of speech more or less imperfectly. Permanent aphasia practically never occurs.

As a rule then there is, after the convulsion or series of convulsions is over, paralysis of one side of the body affecting face, arm and leg. The facial affection is usually slight; rarely permanent. The arm is generally more affected than the leg; the knee-jerk is exaggerated, and ankle clonus is of frequent occurrence. Rigidity is not usually so extreme as in adult cases; but some features exist in infantile hemiplegia which are at any rate much less common in adults. Amongst these probably the most striking is what is sometimes spoken of as atrophy of the affected side. But this is obviously an erroneous term to use, the condition is more correctly described as maldevelopment of the affected side. In a large proportion of cases the smaller size of the structures on the affected side is striking. This defect in development affects not only muscles and other soft structures, but, in a well-marked case, the bones also are much smaller than the corresponding structures on the unaffected side. The arm is usually more strikingly affected than the leg; and this is in accordance with the usual distribution of the paralysis. Not infrequently when the defect in development is so slight as to be doubtful, a comparison of the two shoulder-blades will make it evident that one is distinctly larger than the other. In rare cases only is the difference in size between one side of the face and the other distinct and striking. The dependence of this feature of infantile hemiplegia on the central lesion is probably indirect. The defective development of the structures on the affected side is more probably the result of the disuse which the paralysis enforces, than of any direct neurotrophic influence.

Another condition found in infantile hemiplegia, not so frequently as to be pathognomonic but much more frequently than in other varieties of hemiplegia, is a form of tremor, the so-called mobile spasm, or *athetosis*. This only occurs in a small proportion of cases of infantile hemiplegia (probably about 10 per cent); but it is much more frequent than in hemiplegia occurring in the adult. Indeed, its occurrence in adults, unless the hemiplegia date from an early age, is very uncommon; and in most of the cases in which its onset has taken place during adult life it has been in patients in whom the paralysis was the result of accident. Its presence may lead to hypertrophy of certain arm muscles. This form of movement has been already described under birth palsy. In that section also a curious form of tremor, sometimes named "chorea spastica," closely simulating the tremor of disseminated sclerosis in that it is evoked only during voluntary movement, has been mentioned. Of forty-two cases of infantile hemiplegia two manifested this peculiar tremor; but probably it does not occur nearly so frequently as this proportion would seem to indicate.

As I have said, this condition is usually ushered in with a series of fits beginning on the side subsequently paralysed. Frequently these fits recur at intervals, sometimes short, sometimes prolonged. They are, as a rule, unilateral, affecting the paralysed side. Some observers, however, have stated that the fits affect the non-paralysed side. It is conceivable that this may be the case if the paralysis is severe; while the paralysed side may be that affected in the fits only when the paralysis is less complete. The fits are usually accompanied with loss of consciousness; and consist of tonic and clonic spasm, with occasional tongue-biting, and relaxation of the sphincters. But sometimes the attacks are of the nature of petit mal, and consist in momentary loss of consciousness without convulsion. The cases in which this occurs are cases in which the paralysis is slight. In one the attacks, which occurred frequently, were succeeded by the condition known as automatism, in which the patient, while still unconscious, performed apparently purposive acts. Such a condition is, of course, not only of great interest, but of the highest medico-legal importance. [See later art. on "Epilepsy and Insanity."]

Besides the evidences of interference with physical functions, in a certain number of cases there is some mental deficiency. Often this is considerable in cases in which the physical functions are but slightly interfered with; and in such cases the mental disturbance not infrequently assumes the form of restless and irresponsible activity. In other patients a dull and apathetic condition of dementia is present; and in them the physical condition is frequently one of considerable, sometimes profound, unilateral weakness. But no constant relation is to be found between the physical and mental impairment, and where the latter exists there is probably mischief in the præfrontal region. It is easy to imagine a condition in which it would be considerably affected without any very noticeable degree of interference with motor functions.

Besides these cases of what may be called infantile hemiplegia proper, other cases of hemiplegia of cerebral origin are met with in children, occurring after acute diseases; especially scarlet fever and diphtheria. Such a condition is the result of some intercurrent complication of these diseases. In the former disease it is probably due to embolism, a result of associated endocarditis; in the latter the same process has been alleged, but in four cases seen by myself there was no evidence of any endocarditis; and thrombosis or hæmorrhage is to be regarded as a more likely cause. Hemiplegia also sometimes follows typhoid fever in a child. As blocking of veins in other parts is not uncommon in this disease, a similar process in the cerebral veins is to be regarded as the probable cause of the palsy. The hemiplegia in the cases of this nature is not generally associated with fits, nor with any profound psychical change; but is accompanied by maldevelopment of the limbs on the affected side. In regard to diagnosis, prognosis, and treatment, what is to be said in reference to ordinary cases equally applies to cases of this nature.

Pathology and Morbid anatomy.—The ultimate pathology of this condition is still largely a matter of conjecture. There does not seem to be any constant relation of season to the onset, as is the case in Infantile paralysis; but it must be remembered that these cases are usually seen when the paralysis has already existed for years, and when the recollection of the conditions attending its onset has become indistinct and untrustworthy. Even the morbid anatomy is still uncertain, for few patients die, or at any rate are examined after death while the condition is recent; and the state of the brain after a time is such as to give little or no clue to its initial morbid state. Two views are held with regard to its morbid anatomy; according to Strumpell the condition of infantile hemiplegia is a result of an acute inflammation of the gray matter of the cortex, analogous in some ways to that in the gray matter of the spinal cord which underlies acute anterior poliomyelitis; this condition he names *Acute polioencephalitis*, and if his view be accepted the ultimate pathology would probably have to be looked for in some infective process. The other view regards the condition of the nervous system as secondary to some vascular obstruction; and this, according to Sir W. Gowers, is to be looked for in the veins of the surface. No definite data are yet available to decide between those rival hypotheses. The condition which has been found in the cases hitherto examined has been one either of cystic formation (*porencephalus*) with much atrophy of the affected hemisphere and of the correlated parts of the nervous system, or one of sclerosis of the hemisphere; and both these conditions are, of course, compatible with the initial lesion formulated in each hypothesis. Nor can anything definite be said as to the cause of the athetosis present in a number of the cases. It is probably a result of imperfect destruction; but whether in the cortex or in the deeper structures it is still impossible to say. Even the case published by Beevor and Horsley, in which removal of a cortical area in an adult brought about a cessation of the athetosis, is not conclusive.

The **diagnosis** is not ordinarily difficult. A case of birth palsy may closely simulate the clinical condition of infantile hemiplegia; but the history of weakness actually at the time of birth, although only first noticed a few days later, will usually be sufficient to indicate the category to which a given case is to be referred. From infantile paralysis the state of the reflexes and the absence of any electrical change in the muscles of the affected limbs will sufficiently serve as a distinction. Some cases of myopathy may present a superficial resemblance; but the hemiplegic character of the affection, and the presence even of excess of the knee-jerk, as well as the history, especially of convulsions, will usually be sufficient indication to allow of a correct diagnosis being made.

Apart from the danger which the weakness and the liability to convulsions imply, the **prognosis** is good, so far as life is concerned. It is also found that the hemiplegic weakness is usually greater immediately after the onset of paralysis than it is ultimately; so that we may predict a certain degree of improvement. But the weakness will remain: the child will probably be backward, and may have a definite degree of mental weakness. The fits can usually be controlled if the patient is carefully, judiciously, and persistently treated. This, however, is only partly true of cases in which petit mal is present. But even in cases in which no fits have occurred, except the one convulsion or the series of fits at the onset, it must always be remembered that fits are prone to occur, although their occurrence may be delayed even for as long as several years. If speech has been impaired the impairment is rarely if ever persistent, and sensory impairment is not found, although hemianopsia has been described.

Treatment.—Great care and patience must be exercised in cases of mental backwardness to develop the existing mental power cautiously and judiciously. The same is true of the defective physical power; and mild gymnastic exercises and passive movements will do much to increase the power which the patient retains. Massage will also assist in minimising the rigidity; but electricity is not of much use, and its application may do harm by causing an amount of discomfort and alarm quite out of proportion to any benefit likely to be derived. Convulsions will be treated like epileptic fits. They are usually amenable to ordinary treatment by bromides; but the attacks are all or mostly attacks of petit mal, which are much more intractable. Fresh air, good food, and the best possible hygienic conditions are, of course, essential.

MICROCEPHALY.—This condition deserves a brief notice. Cases of different kinds have been gathered under this name, but, so far as can be ascertained, only two varieties need be considered; namely, those in which the small or deformed skull is secondary to actual brain disease, such as the birth palsy, or infantile hemiplegia just considered, and those in which the smallness of the skull and of the body generally is but a local expression of a general developmental defect in which the brain itself shares. The cases of the former class may be characterised by some local weakness or paralysis; in those of the latter the weakness is universal: in both classes the mental state is very backward. In the last few years operative treatment has been recommended in such cases, the object being by removal of the cranial wall to relieve a hypothetical condition of pressure and consequent interference with brain growth. No results, however, have been obtained to justify such procedures, and the cases cannot be regarded as in any degree amenable to treatment other than the educative and disciplinary influences resorted to in the treatment of weak-minded children.

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J. T.

OTHER DISEASES OF THE NERVOUS SYSTEM

DISORDERS OF SLEEP

INSOMNIA—SOMNOLENCE—DREAMS AND NIGHTMARE—NIGHT-TERRORS OF CHILDREN—SLEEP-TALKING—SLEEP-WALKING.

Physiology.—It is unnecessary in this article to enter into a discussion on the physiology of sleep. Certain symptoms—diminished metabolism, shallower respiration, less frequent pulse, diminished secretory activity, and loss of consciousness—are recognised as constant accompaniments, but a satisfactory explanation of the condition is not yet found. There are several hypotheses as to the causation of sleep. It has been attributed to cerebral anæmia; to chemical changes in the brain cells or neurons, such as an exhaustion of their intramolecular oxygen, or an accumulation of fatigue products; to a contraction of the dendritic processes, and a consequent break in the transmission of nervous impulses; to an expansion of the neuroglial cell processes insulating the nerve-cell processes, and producing the same effect; and to a purely psychological condition, namely, loss of consciousness apart from any physical or chemical change. The last explanation is simply a cloak for our ignorance. The most probable hypothesis is that of an altered metabolism of the cerebral cells dependent upon exhaustion and diminished influx of stimuli.

Anæmia, or relative anæmia, is an important factor, according to many authors the most important. As the vascular contents of the cranial cavity must remain practically constant, any loss of blood in the arterioles and capillaries must be compensated by some increase elsewhere. According to Cappie, this occurs in the pial veins; according to Howell, in the veins at the base of the brain. In any case there is a relative anæmia—a diminished blood-flow through the cerebral cortex, and consequently a diminished metabolism and depressed function; but the bearing of this upon the production of sleep we do not know. Howell believes it is important. From plethysmographic observations on the cutaneous circulation, he was led to suppose that exhaustion of the vasomotor centre is the all-important condition; but this opinion has been strongly criticised by Dr. Leonard Hill. As far as we can see, neither the circulatory, nor the histological, nor the psychological hypotheses are sufficient to account for sleep; and, with the exception of the circulatory, none of them bears any practical significance. The chemical hypothesis, on the other hand, is of direct importance, as it explains in some measure why, in order to induce sleep, it is necessary to rid the organism of fatigue products and toxins.

Amount of sleep.—The amount of sleep required by a healthy person depends upon his age, the quality of the sleep, and his idiosyncrasy. A baby sleeps the greater part of his time; as the child grows up less sleep is needed; and at the age of fifteen, nine to ten hours are usually thought enough (*vide* vol. i. p. 470). Between fifteen and twenty-five years of age eight hours should be allowed; afterwards the amount must depend upon the work and requirements of the individual. Some people can do with from four to five hours; and others with even less than this amount, as was the case with Brunel and Alexander von Humboldt; for the majority of people, however, from six to seven hours are usually enough. Idiots and persons of weak intellect often sleep excessively, and even in more normal individuals a condition of abnormally prolonged sleep is occasionally recognised. Old people also require an increased amount of sleep. When sleep is profound and continuous, less is required than when it is broken and superficial.

INSOMNIA.—By this term is meant loss of the normal amount of sleep. It includes all grades, from simple restlessness to total loss of sleep. From what has been said it is obvious that the term is comparative; that what is sleeplessness in one person is not so in another. Furthermore, the kind of sleeplessness varies. Some people go to sleep directly after getting into bed, but awake every hour for several hours, sleeping well the rest of the night; others, especially the gouty, awake punctually at three or four, and are unable to sleep again, or, at any rate, until it is time to get up; others again find great difficulty in getting off, but, once asleep, do not awake until the morning. Some persons, as we have said, do not sleep at all, and these cases are of serious omen. It has been stated that sleep is more necessary than food; that animals die sooner from sleeplessness than from starvation; yet patients occasionally assert that they have not slept for weeks. In most cases this is probably illusive: they sleep without realising it.

In general, sleeplessness cannot be considered a disease in itself; it is, however, an important symptom of many diseases, and in certain cases, especially of neurotic women and over-tired men, it is the predominant one, and calls for careful treatment, otherwise it may lead to grave consequences. As the essential feature in the treatment of this condition is to discover, and, if possible, to remove the cause, it will be convenient to consider insomnia with regard to (*a*) its causes, (*b*) its treatment.

Causes.—These are many, hence it is necessary to adopt some principle of classification. This has been done by most authors, but no system has yet received general acceptance. Germain Sée recognises nine divisions: doloreus, digestive, cardiac and dyspnœal, cerebro-spinal and neurotic, psychic, insomnia of cerebral and physical fatigue, genito-urinary, febrile, and toxic. Others admit only four or five. We shall adopt a division into four. But it must not be assumed that all causes can be uniformly arranged in these classes; in some cases the immediate cause will fall under two or more divisions. Such divisions are of course only justified for descriptive purposes.

1. *Irritative causes.*—This class includes all forms of insomnia caused by pain and like uneasiness. In children, teething, indigestion, worms, and so forth, are frequent causes. Eye strain—as in hypermetropia and astigmatism, the irritation of an eczema, the inconveniences produced by faucial adenoids, and other similar conditions, are also etiological factors of insomnia. After surgical operations, even in the absence of pain, insomnia is not infrequent; this may be due in part to the constrained position of the patient and some irritability of the wound, in part to toxæmic and psychical causes. Cold feet, vesical affections, asthma, bronchitis, and other diseases accompanied by troublesome if slight irritations, for example, cough, or pruritus (local and general), also fall under this head. But the most important causal factor of this group is undoubtedly pain. Of all diseases accompanied by pain, as neuralgia, sciatica, migraine, angina pectoris, aneurysm, locomotor ataxy, the various forms of colic, acute inflammation of the serous membranes (peritonitis, pleuritis, pericarditis, meningitis), cerebral tumours and abscesses, malignant growths in various parts, diseased bone, acute and chronic rheumatism, osteo-arthritis, syphilis (the pains of which are often worse at night), and acute gout, sleeplessness is a symptom. The insomnia of all or many of these may involve other etiological factors, but the pain is the predominant one, and that which necessitates treatment.

2. *Toxic causes.*—A large number of diseases are due to the presence of some toxic agent in the blood, or are accompanied by it; and such diseases are often attended by troublesome insomnia. Alcoholism and nicotism, the exanthems (measles, small-pox, scarlatina), enteric and other continued fevers, erysipelas, diphtheria, pneumonia, influenza, hepatic disorders (the lithæmia of Murchison), dyspepsia and other gastric and intestinal disorders, rheumatism, gout, and Bright's disease are some of the conditions which fall under this head.

The insomnia accompanying heart disease and altered vascular conditions (arterio-sclerosis, etc.) may also be included in this division, as their origin in most instances appears to be a blood poison. This form of insomnia also is due in part to a deficient blood-supply, and possibly to an altered cardiac action. The sleeplessness induced by certain beverages, such as tea, coffee, and cocoa, requires mention here. These, especially if taken strong and late at night, are in most people powerful preventives of sleep. Strychnine and other nervine tonics, when taken regularly, often cause sleeplessness during the earlier part of the night; but in certain conditions of exhaustion, by improving the circulation and other functions, they prove excellent hypnotics.

Insomnia is also a symptom of certain drug habits, such as opium-eating, morphinism, and cocainism.

3. *Psychical causes.*—Grief, shock, worry, and mental anxiety are among the most frequent causes of insomnia. In many persons a predisposing factor—a nervous temperament, neurasthenia, hysteria, hypochondriasis—also exists, and among such persons insomnia may easily be established as a habit. In students, overwork, especially if accompanied by irregularity in retiring to rest and the prospect of an examination, are the most frequent causes. Women at the menopause often suffer from insomnia, partly, perhaps, owing to the accumulation of toxic products not eliminated by the catamenia. The various forms of insanity—such as mania, melancholia, general paralysis—are accompanied by insomnia: in some cases it appears as a premonitory symptom; although there can be no doubt, on the other hand, that continued loss of sleep is also a factor in the production of mental aberrations. Cases of severe chorea and of paralysis agitans are also marked by sleeplessness.

4. *Causes arising from change in the mode of life.*—Eating late dinners by those unaccustomed to them, and change of climate, especially to high altitudes, or in some individuals even to the seaside, sometimes give rise to temporary insomnia. Nurses who have been on night duty, after changing to the day frequently suffer from sleeplessness; and the same condition occurs in other people with intermittent nocturnal occupations. Sometimes very simple changes in the mode of life of individuals will lead to insomnia. A patient of mine and his wife both slept very badly for the first three months after beginning to cycle. This may have been due to excessive fatigue, or possibly to increased waste products in the blood, or to both combined.

Pathology.—Upon this subject we know very little; and so long as the physiology of sleep is ill understood, the pathology must remain unknown. As we have seen, the condition of the circulation and the composition of the blood are important factors in the production of sleep, and when the brain cells are improperly supplied with blood (as in anæmia and heart disease), or with impure blood (as in the various toxæmic diseases), we can readily understand that the nutrition of the nerve-cells is altered, and that some deviation from normal sleep must occur.

Treatment.—This naturally falls under two heads—general or non-medicinal, and medicinal—although these cannot well be separated in practice. In all cases the first thing to look for is the cause, so that, if possible, this may be removed. If it be an indigestion, this should be treated. Acid dyspepsia and that attending gout are frequently accompanied by insomnia. In these cases, careful attention to the diet, the avoidance of tea and fruit (cooked and uncooked), and of all substances containing vegetable acids, except in so far as these may be used in treatment, are essential. Fruit, owing to the acid it contains, is, I believe, an unsuspected cause of indigestion, gout, and sleeplessness in many persons: so also are acid wines. For gouty persons much butcher's meat, especially beef, should be prohibited, and white fish, poultry, and game ordered in its place. Fried fat, such as that on the outside of fried fish, should also be avoided. An alkaline bitter before meals, an occasional mercurial at bedtime followed by a saline aperient the next morning, and, for the gouty, colchicum or sodium salicylate should be prescribed. Sometimes a dose of Gregory's powder at bedtime, or a mild carminative, is of value. An overloaded colon is a common cause of sleeplessness in elderly people; for this an evacuation of the bowels at bedtime is the best means of relief. One of my patients found that a little peppermint water taken at bedtime gave him a better night than any hypnotic. In the insomnia of the aged and the overtired, and in that due to cardiac and vascular disease, strychnine and nux vomica are beneficial remedies: they tone up the circulatory system, remove indigestion and flatulence, and act directly on the cerebral cells. In cardiac cases and in neurasthenics with low arterial pressure, digitalis and the other cardiac tonics may be resorted to. In anæmia, iron, nux vomica, and sometimes digitalis are required; and similarly other diseases which are causal factors of insomnia must be treated in a more or less specific way. The appropriate remedies for each disease are described in other parts of this work.

In all cases of insomnia certain general precautions must be taken. The bedroom ought to be in a quiet part of the house, well ventilated, and of moderate temperature. Light should be carefully excluded, and the apartment scantily furnished. The bed should vary according to the habits of the individual; for young and middle-aged adults a firm mattress is the best, but for the old a softer bedding may be necessary. The covering should be light and warm; but in the use of pillows no general recommendation can be made. Some people sleep better with the head raised, others prefer to keep it on a level with the body. In heart disease it will be found necessary to raise it; but as a rule this precaution may be left to the choice of the individual. For broad-shouldered people Whitla recommends the wedge-shaped pillow used by the Germans. Invalids find much refreshment in the ultimate use of two beds, for day and night.

For ordinary cases of sleeplessness simple means very often suffice. Some people read themselves to sleep, some count, others, like Southey, think of some monotonous discourse. One of my patients used to hang his feet out of bed for some time and then put them in again. Walking about naked, or a cold or tepid bath is often useful. Massage, especially of the abdomen, thighs, and legs, as in Dr. Eccles' method of treating insomnia, is sometimes advantageous. This method is believed to produce temporary anæmia of the brain, by causing a determination of blood to the manipulated parts; and it may be further aided by a hot compress to the abdomen. In the case of cold feet, vigorous rubbing, or a hot bottle, or a hot footbath with mustard in it, is beneficial; or again a hot sitz-bath may be used. Attention should also be given to the work of the stomach. As a rule a light supper is the best, and for many, and especially those who awake in the middle of the night, a little hot milk or meat juice containing a small amount of alcohol is helpful. The evacuations should also be attended to, and the bladder especially should be relieved.

Sleeplessness from overwork, especially from literary work, requires mental rest and change of air and scene. Temporary exposure to the cool air of the bedroom, or the wet pack, or a bath is often of use; but if the insomnia continue, it is necessary to give a mild hypnotic, such as twenty grains of sulphonal or trional, or thirty or forty grains of bromide of potassium, to break the habit of sleeplessness. Capsules containing ♏xxx. of turpentine given at bedtime are sometimes beneficial in the insomnia of overwork and worry. The drug acts as a stimulant and derivative, and is stated to succeed best in plethoric cases. No beverages containing caffeine should be taken after breakfast.

In nervous and hysterical women, and especially in women at the menopause, the bromides are very useful. I have long been in the habit of giving a mixture of bromide—either of potassium, sodium, or ammonium—tincture of sumbul, and tincture of hop, in camphor water, at the climacteric; and it has helped to remove the insomnia as well as the mental depression and flushing heats so common at this period.

The sleeplessness of the insane requires careful management. In the early stages of acute mania the bromides, chloral, hyoscine hydrobromide, and other sedatives are useful; but a hot bath at a temperature of 104° F., and cold water simultaneously poured upon the head are most efficacious in inducing sleep. In melancholia, where arterial pressure is usually high, paraldehyde in doses of ♏xl. to ♏xc., or even more, is a valuable hypnotic; so is morphine; but a 1-grain dose of erythrol tetranitrate, by reducing blood-pressure, will frequently act better than anything else.

In mild cases of delirium tremens sleep usually comes on after a time, whatever treatment be adopted; in the more severe cases chloral and bromides, alone or in combination, are beneficial. Paraldehyde is recommended by some physicians. Opiates may be given, but in most cases hyoscine is probably a more efficient remedy. Among the medical officers of the United States army 20 grains of powdered capsicum in the form of a bolus is the favourite hypnotic in this complaint. I have no practical experience of this prescription, and cannot therefore express any opinion of its value as a mode of treatment. Cerebral depressants should be given as little as possible, and the treatment should be confined chiefly to feeding and tonic measures.

In pneumonia sleep comes usually at the crisis; but where this has not occurred I have occasionally seen a hypnotic, such as chloralamide or paraldehyde, turn the scales in favour of the patient.

In pleurisy, and most other serous inflammations, 5 to 10 grains of Dover's powder usually conduce to sleep; mainly by relieving the pain. A hypodermic injection of morphine may be given with the same object in view.

In bronchitis, chloral and chloralamide are safe hypnotics; as a rule opiates are to be avoided.

The sleeplessness of asthma is relieved by remedies which cut short an attack, such as chloral hydrate, the fumes of Himrod's and other anti-asthmatic powders, the hypodermic injection of morphine, or, in some cases, a dose of 5 to 10 grains of citrate of caffeine. Bromides are also useful and so is paraldehyde, which both relieves the asthma and causes sleep. A change of locality, if only to another part of the same town, often succeeds. In one case, the removal of a student from Downing College, Cambridge, to a house across the street brought relief; and in another of my pupils the change from Caius College to a house in another part of the town brought to an end a most troublesome attack of asthma.

The insomnia of heart disease is benefited by digitalis, strophanthus, strychnine, and other cardiac tonics; but in some cases it is necessary to resort to morphine, either by the mouth, or still better, hypodermically, as first suggested by Professor Allbutt. Paraldehyde and chloralamide are most useful in my experience; they are less depressing to the circulation than chloral hydrate. Ice to the head is recommended by Dr. A. Morison, where the vital forces are not too low, or the temperature subnormal. It often produces sleep rapidly, with a more regular cardiac action. Heat may possibly answer in other cases presenting a subnormal temperature.

In chronic Bright's disease insomnia is occasionally very troublesome. Eliminants, such as aperients, should be tried, and if they do not succeed chloral hydrate may be given; it is a safer drug in kidney than in heart disease, the accompanying reduction of blood-pressure being usually beneficial. Morphine and hyoscine hydrobromide subcutaneously injected have been recommended in obstinate cases; but their employment requires great caution. Erythrol tetranitrate, by reducing blood-pressure, often acts like a charm even when sedatives have failed; and in one of my patients thorough rubbing of the skin by means of a flesh-brush induced sleep, and very materially relieved the restlessness of this complaint. In the sleeplessness dependent upon cirrhosis of the kidney Dr. Nestor Tirard recommends hyoscine, sulphonal, and paraldehyde; he is opposed to the use of opium and morphine.

When pain is the causal factor of insomnia morphine is the best general remedy, and it should be pushed until relief is obtained. In cases of neuralgia, locomotor ataxy, and so forth, some of the synthetic analgesics—phenazone or phenacetin—are of value. These drugs act also, I believe, as hypnotics in cases where there is no pain.

Calcium chloride is a valuable remedy in the insomnia due to pruritus.

This article would be incomplete without a more detailed reference to some of the more important hypnotics. These are comparatively few, although the number of the drugs of this class which have been advocated from time to time is large.

The *bromides* of potassium, sodium, and ammonium are mild, safe, and trustworthy hypnotics. They tend to depress the functions of the spinal cord, but their effect upon the heart, lungs, and other organs is small. Potassium bromide is the most depressing of the three, ammonium bromide the least; but in therapeutic doses this action is not very obvious, and the potassium salt is the most convenient to use. By many it is regarded as the most trustworthy.

Various other bromides also—for example, lithium bromide and hydrobromic acid—have been given as hypnotics; but none of these presents any material advantages over the salts more commonly used.

The bromides are useful in cases of insomnia due to worry or overwork. When relief cannot be obtained by non-medicinal means they are valuable to break the habit of sleeplessness, and thus to restore the brain to a more normal condition. In the presence of pain, however, they are generally useless. They are also the most useful drugs for repeated administration, as they appear to produce less serious ill effects than other remedies of this class. The dose may be given just before retiring, or, as recommended by Macfarlane, in two or three doses during the latter half of the day.

Chloral hydrate is a more powerful, but also a more harmful drug than the bromides. It produces general nervous depression, affecting both the brain and spinal cord; and it is also markedly depressant to the heart and vascular system. On this account it should be avoided in all cases of heart disease with symptoms of heart failure, and generally in diseases with a low blood-pressure, such as enteric fever. In cases of high arterial pressure, however, it may usually be given with impunity. Also in the more simple forms of insomnia, when bromides prove insufficient, to combine them with chloral hydrate is judicious and often successful. It should, however, be used with care, both on account of its circulatory effect and its tendency to form a habit. A suitable dose is 15 to 20 grains, or, combined with bromide, 10 to 15 grains; but sometimes more is required. Large doses should, however, be avoided.

Butyl-chloral will frequently relieve the insomnia due to neuralgia of the fifth nerve.

As substitutes for chloral, certain derivatives, purporting to possess its useful properties without its ill effects, have been recommended. Of these two only, chloralamide and chloralose, are of practical importance.

Chloralamide, or chloral-formamide, is undoubtedly less toxic than chloral hydrate, and does not produce the same depressant effect upon the circulation and respiration; but it is also a less powerful hypnotic. It may be used in doses of 30 to 40 grains in all cases where chloral is indicated, and in some forms of heart disease and bronchial affections.

Chloralose is a more powerful drug than either chloral hydrate or chloralamide. It differs from them in stimulating instead of depressing the functions of the spinal cord; in animals, to such a degree that convulsive tremors are produced. The heart is said to be uninfluenced by therapeutic doses; the blood-pressure is slightly raised. It may be used as a general hypnotic, and is especially recommended in cardiac and digestive troubles. In chronic Bright's disease, and others attended with increased arterial blood-pressure, it is contra-indicated on account of its influence in raising the pressure. Various opinions have been expressed about this drug. Some have found it beneficial, others not; and some have observed spasmodic twitchings from its use. It may be given in doses of from 3 to 6 grains in 1½ to 2 ounces of water a little while before going to bed.

Paraldehyde is a safer drug than any of the preceding. It produces sleep without any distinct respiratory or circulatory effect; but it is slightly irritant to the gastric mucous membrane, and possesses an unpleasant smell, which can be detected in the breath after its administration. On this account, indeed, it is not so likely to cause a habit, although a few cases of this are on record. It may be generally used as a hypnotic, and is specially serviceable in asthma, heart disease, and melancholia. It is best administered with tincture of orange, in a dose of ♏xl. to ♏xc., or upwards. Dr. Clouston has given ʒiv. to ʒvj. doses.

Sulphonal and *trional* are valuable hypnotics; the former in doses of 15 to 30 grains produces sleep in two to three hours, followed by no ill effects. Drowsiness is sometimes experienced during the following day; and the sleep of the succeeding night is often as sound, or sounder, than that of the night of administration. It possesses no action on the circulatory and respiratory systems. As it is slightly cumulative, repeated dosage may lead to hæmatoporphyrinuria and other untoward effects, and on this account it should not be continued longer than from four to five days. An interval of an equal or longer period should be allowed to elapse before resorting to it again, if this be necessary. Owing to the insolubility of the drug in cold water, it is best given in hot fluids, such as milk or soup; in this way a more rapid effect is obtained. Sulphonal is useful in all mild forms of insomnia, and one dose often produces two nights' sleep. It is adapted for continued administration, provided that intervals are allowed to elapse every few days, and that care be taken to avoid constipation.

Trional possesses similar properties to sulphonal, and exerts a similar effect. It is somewhat more powerful, and, being more soluble, it does not lead to a second night's rest, like sulphonal. Its uses are the same as those of the latter drug. It is valuable in the insomnia of children. Some authors say that it relieves pain as well as induces sleep.

Alcohol, in those unaccustomed to its use, is a valuable hypnotic in mild cases; especially in the form of beer, stout, or whisky, at bedtime.

I have ceased to use urethane as a hypnotic, as it is very uncertain in its action; and chloral-urethane or ural—introduced by Poppi—has no advantages over chloral, as in like manner it lowers blood-pressure.

Opium and its chief alkaloid *morphine* are the most useful hypnotics where there is great pain, and only in a few other cases should they be used; for instance, in recent and acute insomnia from worry, overwork, or shock. Their influence on the heart and vascular system is small, so that in heart disease, especially if attended with pain, they may generally be employed; but their influence upon the respiration is well marked, and in all respiratory diseases accompanied by impeded respiration from excessive secretion they should be regarded with disfavour. Opium inhibits most secretions, constipation and other undesirable effects usually accompanying its use. Rapid habituation also ensues, and in some patients marked excitement results from its first administration.

In painful maladies morphine hypodermically should be pushed until the pain is abolished; but on account of the well-known toxicity of the drug, care should be taken in its administration. In heart disease, $\frac{1}{8}$ to $\frac{1}{4}$ of a grain of morphine may be given subcutaneously, especially if there is stenocardial pain; or the drug may be taken by the mouth.

Opium is more slowly absorbed than morphine, and its action is correspondingly slower and more prolonged. It also possesses a greater local effect upon the alimentary canal, producing more marked constipation. It may be used to prolong the effect of morphine, or in cases, such as gastric cancer, where a local effect may be of value in aiding its general action.

Owing to the rapid establishment of tolerance, its ill effects, and tendency to induce a habit, morphine is not a good general hypnotic; and many attempts have been made to discover a substitute possessing its hypnotic and analgesic actions without its inconveniences, but so far without success. Certain derivatives of morphine have been suggested; but these are mainly sedative, and are not sufficiently powerful to be used in those cases where morphine is indicated.

Hyoscine, in the form of one of its salts, is one of the most powerful hypnotics we possess, and is said never to induce a habit. It is of special advantage in restless cases, such as the insane, where it is impossible to administer drugs by the mouth and undesirable to give opium. It is, indeed, the best substitute for morphine we possess; rarely producing excitement, and an atropine-like effect occurring only after considerable doses. Owing to its great toxicity care must be taken in its administration; $\frac{1}{200}$ to $\frac{1}{150}$ grain of the hydrobromide, given hypodermically, is usually sufficient, under ordinary conditions, to produce sleep; but in the insane large doses—$\frac{1}{100}$ grain, or even $\frac{1}{50}$ grain—are often required. These quantities should not, however, be given as initial doses. Valvular disease, so common in acute mania, is a contra-indication to its use. A combination of chloral with $\frac{1}{120}$ grain of hyoscine, or with morphine, has given excellent results in cases of obstinate insomnia with excitement.

Hyoscyamine has been used as a hypnotic in cases in which hyoscine is of benefit, but it is not so valuable. It produces more numerous ill effects, and its sedative action is less powerful. The other members of the atropine group are even less useful. Hurd recommends a teaspoonful of the tincture of hyoscyamus at bedtime as a useful hypnotic in cystitis.

Cannabis indica is used by many as a hypnotic. It is, however, apt to produce excitement, and sometimes this is not followed by sleep. It does not produce constipation as opium does, and it is less toxic; in fact no fatal case of poisoning by this drug is known. Its greatest drawback is its inconstant composition owing to its deterioration with age, and so far no official action has been taken to obviate this. Several preparations are on the market. The best are the extractum cannabis indicæ of the British Pharmacopœia, Merck's cannabinon and extractum cannabis indicæ, and cannabinol. A convenient dose to begin with is half a grain of the extract, but with many samples this amount has to be exceeded. There are no special indications for its use. It is given in migraine and neuralgia, and often alleviates the pain of gastralgia in old people; but it is not a powerful analgesic. In other forms of pain it is less valuable. It may be regarded as a general hypnotic except in cases where its excitant action is contra-indicated; and if a good preparation can be obtained, it appears to be a valuable one. It is an ingredient of the much-advertised hypnotic "bromidia."

Pellotine, an alkaloid of a species of cactus (Anhalonium Williamsii), has been recently advocated as a hypnotic. Sufficient clinical data, however, have not yet been obtained to establish its value; and, as far as we can judge, the results of purely scientific investigation do not prove it to be better than other well-known drugs. It has been used in insomnia from various causes. In locomotor ataxy and other painful conditions sleep was produced in some cases, but the pains returned on awaking. No unpleasant after-effects are said to occur, but giddiness, restlessness, and slowing of the pulse are occasionally noted before sleep takes place. The dose recommended is ¼ to ⅓ of a grain, but Jolly states that 1 grain is the proper dose.

Two other agents may be used to induce sleep if the measures previously considered fail; namely, electricity and hypnotism.

Electricity.—Electricity is regarded as a valuable agent by those accustomed to use it, but it is generally recognised that its employment is empirical. In some cases it produces excitement rather than sleep, and it is said "to be largely a matter of experiment as to who will and who will not sleep better after its use." In the majority of cases, however, sleepiness is induced, and, according to Eskridge, the cases most commonly benefited are those in which the insomnia is due "to mental overwork, worry, alcoholism, and supposed hyperæmic conditions of the brain generally." Riggs also states that it is most useful in the sleeplessness accompanying nervous and mental disorders.

Various modes of electrification—general faradisation, galvanisation of the head, cervical sympathetic, etc., and static electrification—are in use. Dr. Lewis Jones recommends general faradisation or the faradic bath, but the majority of electricians are in favour of galvanism. Weak currents (1-3 milliampères) directed longitudinally and continued until the patient feels drowsy, sometimes 15 to 20 minutes, or even 30 minutes, and repeated on alternate days, appear to produce the best results.

In some cases static insulation is preferred, and positive insulation is said to be better than negative (Rockwell). The method as practised by Dr. H. McClure, late of Cromer, is as follows: the patient, insulated on a glass stool, is brought into connection with the conductor of a Carré or Holtz machine. When thoroughly electrified a fine metal point is held opposite several spots on the scalp and forehead, but not near enough to produce a spark. The sensation is as if a light wind or breeze was playing over the part, and sleep is said often to come on while the treatment is being employed (Whitla).

The influence of electrification, in whatever way it is used, is said to be both temporary and permanent. The temporary effect occurs during the first few nights, and is never so great as that obtained from drugs. But the permanent influence is the end to be obtained. As compared with drugs electricity possesses the advantages of being free from ill effects, and the danger of forming a habit, but in some cases it fails to act, and may even aggravate the symptoms; consequently its effects must be carefully watched (Riggs).

Hypnotism.—Cases of insomnia, rebellious to all other forms of treatment, may be treated by hypnotic influence, but only in the hands of a properly qualified and specially experienced medical man. Under such conditions I believe it to be a valuable therapeutic agent.

SOMNOLENCE.—Somnolence, or excessive sleepiness, as previously mentioned, occurs in persons of feeble intellect. Some old people also sleep a great deal. In cases of organic brain mischief, especially cerebral syphilis (Buzzard), and in some epileptic and hysterical persons, somnolence is met with (*vide* art. "Hysteria"). It has also been attributed to the reflex irritation caused by round worms, as the removal of these has caused the somnolence to cease. Some sufferers from anæmia, leukæmia, and myxœdema sleep too much, the last more especially in the daytime.

The sleeping sickness, or negro lethargy of the West Coast of Africa, is, as its name implies, attended among other symptoms by drowsiness, which gradually deepens into somnolence and profound coma (see article on this disease, vol. ii. p. 479).

DREAMS AND NIGHTMARE occur when certain cortical centres are active, whilst others are in abeyance. They frequently arise from indigestion; but occasionally they are the forerunners of apoplexy or insanity. "The recurrence of a peculiar and unusual dream night after night, particularly if it is of a terrifying kind, should always arouse suspicion" (Macfarlane). A patient of mine, an old lady of eighty-one, had a most distressing dream a day or two before an attack of right hemiplegia.

The NIGHT TERRORS of children arise in connection with alarming dreams. They usually occur in neurotic children, from one to four years old, during the first hour or two after they go to bed, when the sleep is most profound. Dyspepsia is the commonest exciting cause. Children troubled with this affection need soothing by a mother or skilful nurse, careful dieting, a stomachic mixture or powder, and a dose of bromide at bedtime for a few nights in succession to calm the cerebral centres of emotion.

SLEEP-TALKING or somniloquy, and SLEEP-WALKING or somnambulism, are states in which the whole brain is not asleep, but certain centres continue in activity. Some persons, whilst apparently asleep, may even carry on a conversation, as if they were awake.

Somnambulism, which has been described as an acted dream, runs in families, and occurs about equally often in the two sexes. The subjects of it can perform extraordinary feats, such as unlocking doors, walking in dangerous places, as on the edge of a precipice in the dark, scaling a partition of considerable height (8 to 10 feet) between two sleeping-apartments; and they usually do all this without injuring themselves.

It is necessary in some of the worst cases to keep the patient in a room locked from the outside, to fasten all windows, and occasionally to chain one leg to the bed, so as to wake him if he begins to walk. The general health should, of course, be carefully attended to.

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EPILEPSY

The Name.—Epilepsy is a disease of which the chief feature is the recurrence of sudden brief attacks in which consciousness is lost, and with it the control of the muscles. In consequence the patient falls, and the malady received from our ancestors the apt symptomatic name of the "falling-evil" or "falling-sickness." But the muscles commonly pass into a state of involuntary contraction, sometimes slight, but generally intensely energetic, fixing the frame in some strained posture, stopping the breath until the aspect is that of strangulation, and passing away in violent shock-like spasms which leave him in a state of unconsciousness with life only not extinguished. To the Greeks it seemed that the attack must be the work of some unseen agent of demoniacal ferocity and strength, and hence they gave the disease the name of epilepsia, a "falling upon," as by a spiritual agent. This involved a theoretical pathology which was apparently absent from the simpler Anglo-Saxon conception. The same idea, however, is preserved to us in the popular designation "a seizure," and is not quite absent from the name applied so widely in the description of diseases as "an attack."

Definition.—More careful observation has shown that neither fall nor convulsion is a predominant or even dominant characteristic of the disease, common as each feature is. It has shown also that the attacks which constitute the malady present variations so great, both in degree and in character, as to baffle every attempt to frame a clinical definition. The only definition which embraces all forms is "recurring attacks, sudden and very brief, of disturbances of some of the cerebral functions, acting on consciousness, which are not due to a cause outside the brain." But this definition is at once too wide and too narrow. It would exclude ases in which local spasm continues for hours, and it would include nany cases which must be classed as pure vertigo and not as epiepsy, while it would not include the cases in which convulsions are he result of a local injury to a nerve, cases which are, it is true, rare nough.

A nearer approach to adequacy may be found in the pathological lefinition that epilepsy is recurring sudden brief discharge of nerve nergy in some part of the cerebral cortex, not due to the normal cause f such discharge. Exceptions may be taken even to this description, nd their character will appear in the sequel. A definition of epilepsy vould not indeed be worth even this slight attempt, were it not that the ailure may make some facts, to be mentioned presently, more instructive. The only use of defining is to enable us to see better what is on each side f the line. But Nature, when she can, defines a country by a winding iver or a range of mountains. We like to ignore all this, and draw traight lines.

Pathological definition.—Accordingly it may be worth while to see vhat the pathological definition marks off. The sudden discharge of ierve force, so conspicuous in a convulsion by the muscular spasm it roduces, may be traced also in all the minor forms of the attack. In he sensory centres it acts, as we say, "upwards," so as to influence onsciousness only, and may cause, for instance, an appearance of bright tars. When the attack consists of loss of sight, or simply of loss of onsciousness, there is an apparent exception. We associate "discharge" vith over-action. But over-action in one part may arrest action in nother.

The sudden liberation of nerve energy in the gray matter of the erebral cortex occurs in epilepsy without the normal stimulus. There s much in this statement that will render the facts of the disease more ignificant, although it is only possible to glance very briefly at that which underlies it.

The function of the nervous system, and especially of the cortex of the brain, depends upon a capacity for instant release of nerve energy. The instantaneous reflex action, the instant bound away of a frightened animal, prove that the gray matter must hold nerve force almost released. Released from what? We must regard the energy of nerve and muscle which is excited by a touch upon the skin as a form of physical energy, peculiar because acting under the influence of life, released and conducted in living tissues. It is probable that the energy from which nerve force is released is latent chemical energy; with this latent chemical energy is inter-atomic and inter-molecular motion, and that nerve energy is a form of transmitted motion by transmitted chemical processes. The reasons for thinking this I have stated elsewhere (1).

The point for us now is that the nerve force must be ready for instant release in the gray matter. Whatever its source, it must be held in a state of most delicate equilibrium by the restraint which keeps it unreleased but ready. Indeed, there is good reason to think that, to use a simile, the vessels which hold it must be not only filled to the brim and delicately poised, but must be actually overflowing. Both the facts of the constant process of nutritional change, and the evidence of constant activity in the nervous system, lead to the conviction that the readiness of the nerve energy is obtained by its constant overflow, that the chemical processes which induce its sudden generation are always going on in slight degree, and that only thus is it kept so as to be released at once in the needful degree.

But this involves a conception of extreme *delicacy of adjustment* between the chemical relations—the tendency of the atoms to unite in fresh and closer combinations and release their latent energy, that is, their surplus motion—and the attraction which keeps them from so doing. It makes it easy to conceive that the slightest deviation from the normal relation between the atoms and the molecules may bring these into a position in which the equilibrium is suddenly overturned, and a quick combination and sudden release of energy occurs. We can understand that this may be from that marvellous inherited tendency which produces such precision of correspondence between parent and child in the process of nutrition, or that it may be the result of acquired causes, from the sudden shock of a concussion, mental or physical, or of a cerebral lesion which has slightly, but only slightly, implicated the gray matter in a certain part of the cortex. Indeed, when we consider what the state of the nervous system must be in its natural function, we cease to wonder that many persons become epileptic without any traceable cause, and our marvel rather is that all persons are not liable to such fits.

The release is normally effected by a stimulus of a certain character acting in a certain direction among the complex molecules of the nervous system, the arrangement of which facilitates its transmission and action. In the lecture referred to (4) it is maintained that such a stimulus may be best regarded as atomic motion which, added to the pre-existing motion of the latent energy, causes this to form the new and fresh combinations in which there is less constant motion.

A morbid relation of the most delicate and minute character between the molecules may make them susceptible to motion which comes by some other path which we do not discern. Discharge without the normal stimulus may not mean discharge without any stimulus: yet even this is possible; we can conceive that the defective equilibrium may entail a disturbance of the balance, when a certain point is reached, and a discharge which has no other cause than the nutritional augmentation of its own tendency.

The important fact thus is that an extremely slight derangement of the constitution of the nerve tissues may cause a liability to sudden discharges of nerve force, slight and local or severe and wide.

Recurring tendency constitutes the disease.—But another equally important fact must be recognised. The liberation of nervous energy involves the escape of some atoms of the nerve tissue which are at once replaced from the plasma adjacent to them under the mysterious nutritional influence of life. The rearrangement is the same, and yet not quite the same. It differs in just the degree and character to make the same release of nerve energy more easy. Every action paves the way for its own recurrence. The effect is greater the more frequently its occurrence. This is the secret of the acquisition of skill; it constitutes the physical basis of memory; it is this which facilitates the repetition of a given morbid process, and this causes the recurrence of discharge which makes epilepsy a persistent disease. The recurrence of attacks is one great feature of epilepsy, and the recurrence is in great measure the result of the previous attacks. Every fit is in part, at least, the result of those which have preceded it, and in part a cause of those which follow it. Hence the immediate cause of the first fit must be regarded as the cause of the disease, although the essential cause is the disposition, that is, the state of nutrition, which makes such discharge possible. When equilibrium is unstable a trifling influence may overturn the balance, and cause an effect quite out of proportion to the apparent cause. The degree of instability may, of course, vary; and when it is not great the first attack may have been due to some cause of considerable power. Even then, however, we must ascribe the special effect to the special predisposition.

These considerations will make it easier to understand the causes of epilepsy in so far as they are comprehended. But one other important question must also be noticed.

General and local change.—In what is called "idiopathic" epilepsy, that which is largely the result of inheritance, the morbid state consists in some trifling alteration in the chemical constitution of the gray matter in which the instability spoken of consists. But a similar instability may be the result of obvious disease. It is not indeed directly due to disease that can be seen in the damaged part, because for disease to be visible the destruction of nerve tissue must have made even morbid action impossible. But beyond the visible disease, whatever its nature, is a zone of altered nerve tissue in which the slightness of the change permits activity in altered form. In such cases the discharges spoken of may take place. Arising locally, they spread through the brain according to their energy, and then leave a tendency to repetition, which involves parts unaffected by the primary disease. It is not customary to include under the term "epilepsy" active progressive brain disease, such as a tumour, even though it cause epileptiform convulsions. But there is a large class of cases in which an old spot of disease induces such discharges, which may be slight or local at first, but spreading when more intense, and ultimately leaving a general disposition which may be hardly distinguishable from idiopathic epilepsy. Although these cases are quite distinct in primary causation, they are not practically separable from those of idiopathic epilepsy.

Perpetuation of reflex convulsions.—A third group occupies a curiously intermediate position. There are cases in which some local irritation, for instance in the intestinal canal—generally tapeworm—has excited "reflex convulsions," which had been so many times repeated before their cause was removed, that a persistent habit of brain function was established. This may happen even without any inherited disposition. It is possible that some other influences, acting on the nervous system from the outside, may have a similar result. Such cases do not ultimately differ in any important way from the common idiopathic disease, but it is necessary that their initial character should be discerned.

Source of the discharge.—I have spoken of the discharge of nerve energy as proceeding from the gray matter and not from the nerve-cells; and it may be noticed that nothing has been said of morbid changes associated with the idiopathic disease. The two omissions are connected in a significant manner. Up to the present time it has been a fundamental conception that the nerve-cells from which the fibres proceed are the source of the nerve energy which passes along the fibres that proceed from them. Apart from any opinion regarding the electrical nature of nerve force, the obvious analogy between the large cell as a generator and the sheathed fibre as a conductor, has aided in fixing firmly the conception. But the labours of histologists, of Max Schultze thirty years ago, and of Ramon y Cajal, of Schäfer, and others in our own day, coupled with the known facts of physiology, make it necessary to relinquish this simple and attractive notion. We must, it would seem, regard the function of the nerve-cells as nutritional only. On them depends the life both of the processes, short or long, which conduct to them, and the long axis-cylinder process which conducts from them. Through the cell pass the separate fibrils of which the apparently homogeneous axis-cylinder really consists. These arise, without continuity with others, in the spongy gray matter; becoming associated in the "branching processes" they reach the nerve cell to pass through its protoplasm without interruption. We must regard the spongy gray matter as the substance in which the nerve impulses arise under the influence of other impulses conducted to it; and we must therefore look to this, and to this alone, as the place in which any alteration exists that underlies the phenomena of epilepsy. The eyes of the observer hitherto have been fixed on the cells and on the cells alone. We do not yet know how to unravel the complex structure of the spongy substance so as to discern the normal relations and aspect of its constituents. We are not yet on the threshold of the observation of such local disease as must exist in idiopathic epilepsy. Perhaps we never shall be. But it is at least easy to see why we at present know nothing of the pathological anatomy of the malady. Even the aggregations of atoms which form the organic "molecules," under the influence of life, are far beyond our power of observation; so far, indeed, that a vast amount of change, involving extensive and constant functional disturbance, may result from a change in nutrition which cannot be recognised by any means at our disposal. Any perceptible alterations are more likely to be the result of the functional disturbance than the cause of it. Functional action involves local vascular disturbance, and this, if intense, may be manifested by such visible effects as are seen in the medulla oblongata in hydrophobia. But before we can hope to discern that which is connected with the finer disease of the spongy gray matter, we must first become conversant with its minute features under normal conditions.

Causes.—The preceding sketch of the etiology of epilepsy should make it easier to understand the facts of its causation, and should also make it unnecessary to dwell on their significance. The most important impression which the reader should retain from it is that the conditions of the normal action of the brain involve a state of tension ready for release of energy, and a delicate equilibrium which makes the abnormal sudden liberation of energy readily produced, either in consequence of a slight primary perversion of the conditions on which the balance depends, or a sudden and excessive shock to them.

Heredity.—Epilepsy is an inherited disease. Epilepsy and insanity certainly run together in families. The general tendency to one involves also the tendency to the other, although not often in the same individual. In seeking for the family antecedents of epilepsy, it is found that insanity is the only other disease to be taken into consideration. Other neuroses have too many different connections, or these are of so common a kind as to be comparatively insignificant. But if only epilepsy and insanity are considered it is found that in half the cases of epilepsy there is a family history of one or both of these diseases. If the facts be gathered from the lower classes, who seek relief and yield data at the hospitals, this association can only be traced in a third of such patients. But when cases are carefully investigated in private, the proportion rises to one-half: and even this estimate is doubtless below the truth. In the present generation the morbid facts are generally known, but unpleasant events in antecedent generations are often unmentioned in a family, and the knowledge of them may thus be lost.

Of diseases outside the nervous system, it is not possible at present to discern any which stand in a definite relation to epilepsy. Phthisis was once thought to be connected with the disease by some hereditary mechanism, and the cases of phthisis to be heard of in the families of epileptics are many. But the ratio is not found to be larger than in the families of those who are not epileptics, and an investigation from the side of phthisis clearly showed that the apparent connection was due to the frequency of phthisis. In the same manner it is not possible to say whether inherited gout has an influence in the production of epilepsy. It is difficult to resist the evidence of a connection between inherited gout, migraine, and neuralgia; but the connection between these diseases and epilepsy, although definite (at least so far as migraine is concerned), is yet so slight and infrequent as rather to oppose than support the idea that inherited gout is a factor in the production of epilepsy. That the individual subject of epilepsy may suffer more in consequence of the systemic disturbance of gout is of course intelligible without any causal relation.

The consanguinity of parents necessarily intensifies the family habit, but there is no evidence that it has any influence in producing the disease.

Such marriages are indeed far too rare to furnish any material from which an opinion can be formed.

Sex.—The influence of sex at all ages is not great. For every twenty males twenty-one females suffer from epilepsy. This is the result of a careful comparison of nearly 3000 cases, from which all examples of hysteria, not associated with true epilepsy, have been excluded. In some small collections previously published males have slightly preponderated, possibly from the inclusion of cases of syphilitic brain disease.

Age has a great influence on the production of epilepsy. This may indeed be expected from the tendency to spasm and convulsion which is conspicuous in childhood, which lessens as the stability of full development is achieved, and is again increased during the change with which the period of childhood ends. The periods of this change, and of the first three years of life, are those in which epilepsy most frequently commences. One-eighth of all cases begin in the first three years of life, transient infantile convulsions being excluded. Not less than a quarter of the cases begin in the four years, 13, 14, 15, and 16, which, in the majority of cases, include the epoch of puberty. In the second decade, indeed, the malady commences as a continuous disease, in no less than almost half the cases; and in the first decade in more than a quarter: thus three-quarters of the cases of epilepsy begin before 20 years of age. The third decade is responsible for another sixth, leaving only one-twelfth to commence after 30 years of age. But the persistently recurring fits may begin as late as 70 years of age. Yet only one case in 200 commences after 60.

Sex and age.—There is apparently an absolute identity of the two sexes in the early period of embryonal development. There is a similar correspondence between them in their nervous characteristics in the early period of separate existence, especially during the first two or three years of life. Yet the existence of a profound difference in the nervous system of the two sexes is shown by the fact that in cases of epilepsy commencing in the first ten years of life the females exceed the males by 8 per cent, and the difference is greatest in the first year, in which the males are little more than one-half the females. Puberty has a far more profound influence on the nervous system of females than on that of males, and their excess rises, in the second ten years of life, to 20 per cent. With adult life the influence of sex is changed. In the third decennium the disease begins in the same number of males and females. Between 30 and 40 the males are for the first time in excess by no less than 20 per cent. Between 40 and 50 their excess attains nearly 30 per cent. After 50 the same relation persists, but the numbers are too few to permit accurate comparison. Cases of idiopathic epilepsy begin in males up to 75, but almost, if not quite, exclusively in males. We may find an explanation of these facts in the exposure of the nervous system to more constant strain of anxiety in men, but it may also be that some compensatory indisposition is developed in women who have maintained stability through the trying period of development. Certainly facts yield but certain nervous changes may already have taken place, in the higher centres, that had not yet invaded indicating areas; thus the appearance of the disease is sudden, though the detection of the morbid change is late. After the full recognition of the disease, we commonly learn from the friends of symptoms (now appreciated for the first time) which push back the duration of the malady. Such symptoms are the warnings, or prodroma, of general paralysis. These warnings may affect various parts of the nervous system, causing loss of power or loss of control. I will consider some of the more prominent under the heads of mental, sensory, and motor warnings.

On the *mental side* is noticed some impairment of the most specialised accomplishments of the individual; thus the musician ceases to please his trained auditors, the artist's work no longer commands a market, the artisan gets out of work, the soldier loses his good-character badge. There is, in fact, a loss of the highest power of adjustment, a kind of social ataxy which interferes with delicate social intercourse.

Loss or defect in power of attention is very common. Loss of will power, doubt and uncertainty may simulate neurasthenia; and increased irritability, change in temper, abnormal susceptibility to the influence of stimulants of all kinds are of very frequent occurrence. Hysteria and "nervousness" may also occur long before the danger is suspected. Among the *sensory warnings* we may meet with defect in smell, sudden loss of sight of a temporary kind, similar loss of hearing, temporary and local anæsthesiæ, formications, flashes of light, tinnitus aurium, and hallucinations or illusions of one or more of the senses; though in my experience these sensory warnings are not very common. Giddiness and so-called congestions of the brain may occur, or bilious attacks ending in vomiting or fainting fits. If such symptoms appear in patients who seem in other respects likely to suffer from general paralysis, they may be of value as warnings. On the *motor side* there is often well-marked restlessness, or, occasionally, stupor or undue torpor with well-marked sleepiness; slight and temporary aphasia, or loss of power of expression by speech, or by writing, or other defect of the kind, may occur, with some alteration in gait (ataxic or spastic), inequality in pupils, or change in muscular electrical reactions. There may be convulsive seizures or partial palsies, such as ptosis, external strabismus, or loss of power in one limb. Besides these local or special troubles, there is often also a general indescribable change; the person is "not himself," he is tending away from himself. There may be loss of recent memory, inability to transact business, confusion of thought, loss of animal spirits, or, on the other hand, an undue buoyancy with sleepless restlessness. I believe the character of the coming general paralysis is generally foreshadowed by the nature of these earlier warnings, though this is not always the case.

Lastly, I would call special attention to a group of warnings which might be considered with the symptoms of Social Ataxy, but as they have especially important relations I prefer to place them apart. I refer to the *moral perversions* which in not a few cases precede the recognition of general paralysis. Crimes against morality and propriety are the most common, such as criminal assaults, indecent exposure, bigamous marriages, and the like; or, again, stealing, getting money under false pretences, and reckless conduct of business, with apparent fraudulent intent, may be the kind of backsliding. Faintings, bilious attacks, and attacks of "petit" or "grand mal" also occur as warnings.

The invasion may be apparently quite sudden, and it may appear first on the motor, the sensory, or the intellectual side. The first symptoms of the disease may be but the sudden increase of any one of the warnings. A fit of one kind or another may be followed by all the mental and physical signs of general paralysis. An attack of mania, whether this be of the simple emotional or of the delirious form, may pass into general paralysis. Melancholia, or mere silliness, may be the earliest feature of the disease. In rarer cases "folie circulaire" or alternating insanity, systematised hallucinational or delusional insanity seem to initiate the disease; the fact being that the degeneration varies in the part of the brain which is chiefly and earliest affected, and consequently the earliest signs of the disease vary. To sum up: the disease may be recognised suddenly, and any form of mental, motor, or sensory disorder may appear as its first symptom. In some cases the patients seem to have been peculiar for many years, or even to have shown symptoms from early youth which, becoming exaggerated later, prove to be forerunners of general paralysis.

Forms of general paralysis.—Though various authors, including Dr. Mickle, have described certain special varieties of disease in which specific changes in the nervous centres are connected with definite mental disorders, I have not been able to satisfy myself that these divisions can be definitely made. It is convenient, however, to recognise the fact that the symptoms generally combine in one or other of the following clinical groups:—(i.) Acute or galloping general paralysis; (ii.) ordinary general paralysis, with mania and exaltation of ideas; (iii.) melancholic-hypochondriacal or stuporose general paralysis; (iv.) progressive dementia with general paralysis; (v.) general paralysis of the double form; (vi.) spinal general paralysis, that is, beginning with ataxic or spastic symptoms; (vii.) general paralysis in woman; (viii.) developmental or adolescent general paralysis; (ix.) senile general paralysis.

FORMS OF INSANITY IN GENERAL PARALYSIS

	Acute Mania.	Melancholic.	Delusional.	Dementia.	Total.
Bethlem . . .	54	23	3	20	100
Virginia Water . .	39	9	16	36	100
Ticehurst . . .	19	2	...	4	25
Private	100	31	6	63	200

Note.—In the private cases the total number of patients showing degrees of Dementia was 161.

(i.) *Acute or galloping general paralysis.*—The chief characteristic of this disease is the rapidity with which the whole of the symptoms, both physical and mental, may run through their course, and end fatally. The causes of death may have been long at work: signs of mental decay, indeed, may have been passed over as unimportant till the catastrophe revealed them in their true light. The tinder was dry for the spark, and destruction is the result. In some cases death has taken place in six weeks, in others in from three to six months. In one group of such cases acute delirium or acute delirious mania ushers in the disease, and probably some of the fatal cases of delirious mania are really cases of acute general paralysis. A sudden febrile illness, a sudden moral or physical blow, or a fit may start acute general paralysis. Acute delirious mania, or a most remarkable state of restless activity, with some rise in temperature, sets in; the tremor of the muscles about the mouth is generally well marked, the defect of speech is early and well marked, emaciation rapid, face flushed, and the expression anxious; excitement is rapidly followed by mental weakness, loss of vesical and rectal control, bedsores, rapid exhaustion, and death. Fits are not, in my experience, common in this form of the disease, though they may initiate it.

It is interesting to note here that a certain number of patients who have acute delirious mania recover; certain others are left permanently weakened in mind, and a few pass from acute delirious mania into general paralysis. The general paralysis thus started by delirious mania is not always of this acute and rapid kind, but may belong to either of the other forms.

Typical case of General Paralysis.—The following is a specimen of an ordinary case of general paralysis. A married man of about 45, of active habits and with plenty of ability, not belonging to a neurotic family, who in early youth had been a free liver, fifteen years before had contracted syphilis, of which he thought very little, as he had few or no constitutional symptoms. He married when the danger of infection had passed, and for some time he indulged very freely in sexual connection; but he steadied down and was a thoroughly good and respected man of business. Certain severe money losses, due to no fault of his own, worried him, broke down his sleep and appetite, and he sought relief in alcohol. He complained of headache and loss of power, and was regarded as neurasthenic. At this time rest and travel were advised. He was very emotional, even hysterical; at other times he was apprehensive and hypochondriacal. Change and rest, however, seemed to set him up, and in two or three months he returned to work fairly well. He fell down unconscious in "epilepsy"; from this attack he recovered in a few days, but some defect remained in his speech; he hesitated, and did not articulate clearly. Now he took more prolonged rest, and when he came back to town all his friends were struck with his buoyant, exuberant health; and he said he felt better than he had felt for years. This feeling of buoyancy led to amorousness, and not content with home he got into bad company and scandal arose. He rapidly became intolerant of interference, irritable, and passionate. This state of unrest ended in a sudden outburst of mania, with violence and extravagance, during which endless telegrams and letters were sent to distinguished persons to whom he was unknown. Sent to an asylum, he fell at once into all the ways of the place, making himself the agreeable friend of one and all, and never seeming to tire in his work and play. By this time, however, the change in his expression and in his aspect was well marked: his gait was altered, his pupils unequal and sluggish, his writing changed and speech clipped. Every available piece of paper was seized for voluminous scribbling. Still little or no change was seen in his temperature or in his general circulation, though after exercise his breathing seemed to get shorter. He had lost all his finer acquirements, and though he would sing, or play on the piano, his performances were feeble and faulty. Memory was failing, but still was fair; however, he had no power of appreciating his own condition, and still thought himself in perfect health. Sleep was profound and appetite good. This period of excitement lasted for about four months, to be replaced by a phase of general dissatisfaction with his position and surroundings. He complained incessantly that he ought never to have been brought to an asylum, and he appealed so constantly for release that at last this was granted at the wish of his wife. At home he was exacting, irritable and unstable. A change into the quiet country was tried with marked gain, but a return to London was followed by another but much slighter fit which left the mind much weaker, and the patient now exhibited the characteristic "facility." He would read childish novels and cry over them, go out for the same walk daily, and be perfectly satisfied with himself and his surroundings and excursions. Sleep being very good and appetite greedy, he got very fat, his face lost expression, and his skin became sallow and greasy. This calm continued for six months, when a severe fit of an apoplectiform character left him for some days hemiplegic and aphasic. These symptoms slowly passed off, but the man now began rapidly to lose mental and bodily strength; he laughed to himself, was neglectful of cleanliness, occasionally lost control of rectum or bladder, and ate ravenously without mastication; at times he was irritable, but too weak to give much trouble. He got thinner and disposed to bedsores over the sacrum. While trying to get out of his chair he fell, and a rib was fractured, but no complaint of pain was made. Soon after other fits occurred, and for some days he was in a "status epilepticus." One arm was now noticed to be contracting, and he was too helpless to sit up.

The second and third stages had occupied a year; the bed-ridden paralytic state continued for six or seven months, during which his limbs contracted. He seemed quite unconscious of his surroundings; still the organic reflexes persisted, so that anything placed on his lips caused a swallowing movement. Thus paralysed, emaciating, and incontinent of urine and stools, recognising no one, he remained till the temperature rose, with pneumonia at the base of one lung; this rapidly spread, and with a temporary gleam of reason before his death he died quietly at the end of about three years from the time the disease was first fully recognised.

(ii.) *The ordinary maniacal general paralysis.*—It is almost always preceded by a slight period of depression, which often assumes a hypochondriacal aspect. It then resembles ordinary mania, nearly always associated with expansive benevolence with the most wonderful exaltation of ideas. The exaltation is all but universal, so that the patient believes himself to be wealthy, beautiful, a poet, an author, an athlete, and an immortal. Restlessness is also well marked in these cases, but next to expansiveness probably "facility" (as it is called by Dr. Clouston) is most characteristic. The patient, though believing himself to be so mighty, yet, like a child, is very readily diverted from one subject or purpose to another. The mania assumes the emotional or hysterical aspect, and the more marked this is the greater the chance of remission. The second and third stages are alike in all the various forms.

(iii.) *The melancholic form of general paralysis* is not always easy of detection. In many cases, as already said, there is a period of depression which may be of very short duration. General paralysis may be associated with any of the various forms of melancholia. Thus active resistive melancholia, and melancholia with stupor, may be met with. Hypochondriasis is very common in general paralysis, and I have seen melancholic patients with extreme enlargement of ideas—as, for example, to imagine such an occlusion of the bowels that if they burst—as they must—the world would be flooded by the eruption. One such patient believed himself to be a mass of syphilis, and that he would give the disease to hundreds of men in the city. In my experience hypochondriacal notions of bowel obstruction are common in the melancholic forms of general paralysis.

In many cases of melancholia there is great difficulty in getting the patient to speak or to put out his tongue, and consequently it is often hard in the earlier stages to form a diagnosis. Not infrequently I have been misled into thinking the case to be one of simple melancholia till I found the patient, though making constant complaints, was getting fat; then it was found that his pupils were unequal, his speech was defective, and his reflexes abnormal; or perhaps the onset of fits cleared up the diagnosis. Not only marked melancholic symptoms but even "persecution mania" may be an early symptom of general paralysis. I have known one man who with all the physical signs of general paralysis only exhibited simple weakness of mind and the delusion that he had scabies, which he was always anxious to show to all visitors. Another man was very dangerously suicidal because he thought he was infecting the city, and that therefore crowds were waiting to lynch him outside his house. Though suicide is not common in the ordinary general paralytics it may occur in the melancholic and persecuted patients.

It is of great importance in this group to recognise the want of proportion between the bodily and mental symptoms: thus if a man complaining of being miserable, of being obstructed in his bowels, or of being persecuted by his enemies, yet eats well, sleeps well, and gains flesh, he is either a chronic melancholic or possibly a general paralytic passing into dementia. Remissions are less common in the melancholic than in the maniacal form of general paralysis; fits of various kinds may occur, and there is generally a tendency to fatness and dementia.

(iv.) *Demented form.*—Whereas most general paralytics pass ultimately into dementia, some of them pass into weakness of mind from the outset, being from the first dull, heavy, indolent and sleepy. Although they get weaker as the disease advances, yet they rapidly fatten and are very liable to fits. In some the first stage is one rather of mental confusion than of mental defect, but dementia comes on later. Some of these demented cases begin with fits. It has been pointed out elsewhere that the progressive stages of general paralysis are summarily represented in the stages of drunkenness; for alcohol will make one man emotional, amorous, or pugnacious, another sentimental and lachrymose, and a third merely stupid.

It is common to meet with cases of general paralysis of the simple progressive demented type in general hospitals, and many such are treated at home as cases of "softening of the brain." It must not be forgotten, however, that the most placid and docile of patients may suddenly become violent and maniacal.

(v.) *Circular form of general paralysis.*—This is to my mind the most difficult of all forms to detect and to define. It is among those suffering from this form of general paralysis that so many "cures" are recorded; and it was not till I had been misled several times by such cases that the nature of the disease dawned upon me. This variety may be described in a very few words, but the description will not prevent mistakes being made over and over again. A patient (all mine have been men), of the age and with the ordinary history of general paralysis, becomes very extravagant and exalted; he will boast, rush about the town, give presents, make love, drink, and get into the hands of the police; in jail or in an asylum a diagnosis of general paralysis is made, but he slowly improves, expresses gratitude and contrition, and is sent home "well," the doctor believing he has made a mistake in diagnosis. A year later the patient is seen again, but now is in a state of profound melancholia with refusal of food, and the physician feels sure that his original diagnosis must have been wrong. Slowly this melancholia passes off again, to be followed, after an interval of varying duration, by acute mania, resembling the previous attack even in its minutest details; this gives way again to calm, and perhaps to a second "recovery." After the lapse of several years the patient, however, presents the physical and mental signs of general paralysis, of which disease he dies. I do not know any way of avoiding mistakes in these cases beyond careful observation of the physical signs.

(vi.) *Spinal general paralysis.*—General paralysis may follow symptoms of tabes dorsalis which may have lasted for some years without any apparent mental defect; or it may be preceded by marked signs of spastic paraplegia. It is possible that general paralysis may follow disseminated sclerosis; I have seen such cases, but I am in doubt about the affinity of the two diseases. It is thought by some authors that peripheral neuritis —whether due to injury, lead, or alcohol—may give rise to general paralysis; and accordingly a form of general paralysis of an ascending propagated course is described.

(vii.) *General paralysis in women.*—It used to be doubted whether general paralysis ever occurred in women, and it is certain that women of the educated classes rarely suffer from it. I think there is no longer any doubt but that even gentlewomen suffer, though to a much less degree than men. There are no special symptoms to separate the general paralysis of men from that of women. It usually occurs rather earlier in life, it has a tendency to last longer, fits are not so common, and dementia sets in earlier, the patient rapidly passing into a quiet childish state. I think the reflexes, as a rule, are much more exaggerated in women, and it is rare to meet with ataxic symptoms; this is the more interesting when we remember the rarity of tabes in women. Women may menstruate up to a very advanced stage of general paralysis; even in the second stage they may bear children, become pregnant, and be delivered normally. Children born of general paralytic parents (of either sex) will probably show signs of degeneracy; they may be idiotic, imbecile, or vicious.

(viii.) *Developmental or adolescent general paralysis* will be described hereafter by Dr. Clouston. I have seen several such cases, the majority having been taken at first to be cases of disseminated sclerosis, and it was not till later that suspicions of general paralysis arose. This form may occur in children from twelve years upwards; I see no reason why infants should not also suffer. The progress of the disease, in my experience, is not rapid; but dementia appears early, and there is rather a gradual decay than a disorder of the intellect. Paralysis soon occurs, fits are not uncommon, and the patients slowly die exhausted.

I have never yet met with such a case in which there was not evidence of parental syphilis—a very interesting fact in connection with causation of the disease in the adult.

(ix.) *Senile general paralysis.*—This variety is not recognised by many writers, and I am not inclined to regard it as a very distinct form of the disease. I have already pointed out that progressive decay of bodily and mental functions is the essence of general paralysis, and the same may be said of senile dementia; but the latter is rather a normal extinction of the higher functions, while the former is a premature and unnatural decay. It is recognised that some senile persons, mostly men, exhibit a wild, excited, and extravagant train of symptoms, during which they waste their money, go in for sexual debauches, drink, and behave like general paralytics; yet in most of these there is not the universal benevolence which is common in general paralysis, nor is there the marked defect of articulation; the physical weakness is rather paralytic from the first than mere enfeeblement. The memory, too, is more affected in dementia than in general paralysis, and if fits occur they are more probably apoplectic than epileptic. There are cases, however, in which all the symptoms of general paralysis occur in patients over sixty, and such cases must be called cases of senile general paralysis; and I know no limit to the age when true general paralysis may occur, for as age is a relative term so the diseases of old age must be relative also.

AGES OF ONSET OF GENERAL PARALYSIS

	20-30.	30-40.	40-50.	50-60.	Over 60.	Total.
Bethlem	2	49	38	10	1	100
Virginia Water	5	37	46	8	4	100
Ticehurst	4	9	7	5	...	25
Private	42	119	27	12	...	200

Duration of the disease and of its stages. This may vary from a few weeks, in the acute form, to many years in some of the chronic forms, or in those in which prolonged remissions have occurred. I have found the average duration in private cases to be about three years from the first diagnosis. Taking the history of the warnings and the early stages of the disease, I should estimate its duration in private patients of the upper class at about five years from the very beginning to the end. I have known of one or two undoubted cases which lasted from twelve to twenty years. I am inclined to think some well-marked cases never advance beyond the prodromal stage, that indeed there may be an early arrest of the process; and I believe that in occasional cases arrest may occur during either of the other stages; at any rate complete remissions may occur during the prodromal stage, the stage of excitement, or the stage of paresis. I know of no guide to the probable duration of the various stages, and I do not think there is any definite proportion between the length of the several stages. Cases which begin with very acute mania, excitement, and exaltation more frequently present remissions; cases beginning with steady loss of mental power rarely, if ever, have remissions; but the disease may be arrested. In a remission not only is there no progress of the disease, but also a gain of power; in arrest the progress is stopped but no gain is made.

Symptoms.—First I shall take those which may be called the more characteristic, and refer later to the less common symptoms. The only absolutely characteristic symptoms are progressive paralysis and progressive dementia, states which scarcely need any description. On the mental side we find loss of the highest self-control, which is shown in different ways in different persons; there is a loss or defect in the highest and most special acquirements—loss of power of self-judgment, and of judgment in affairs, often engendering extravagant notions of personal worth and power. The emotional instability is also a result of defect of control. Will-power is in defect, so that the patient is facile and easily moved; there is defect, too, in the registration of recent impressions (loss of recent memory).

On the sensory side there is frequently some loss of a special sense, and occasionally hallucinations or illusions; these again may be temporary or recurring.

On the motor side there are defects of facial expression, of speech, of writing, of gesture, and of gait.

On the nutritional side the higher or more organised tissues are replaced by fatty or degenerative products. There is also tendency to vascular and circulatory changes. The blood itself, I believe, undergoes some obscure alteration.

Loss of the highest mental control permits many impulsive acts and emotional outbreaks. An external impression may lead directly to an impulsive act or to a sudden emotional outburst—mere reflex acts uncontrolled and undirected; most of the earlier symptoms depend on this defect of control, which shows itself also in a loss of delicacy in performing the most refined and specialised acts. Tact and accomplishment are lost; the artist is no longer exact in drawing or sensitive in colour; the musician loses sense of time and harmony; the arithmetician is no longer nimble in computation; the clerk is slow and awkward in his writing; the actor loses the versatility and mobility of his features; the artisan is discharged for careless work; the domestic servant gets into disgrace for frequent breakages. The finer defects may run together in several lines: thus the clerk may write badly, speak thickly, and walk awkwardly; and the artist may be deficient not only in his own special art but in related acquirements also.

The defects in the highest control may be progressive, or they may be well marked up to a certain point and then seem to be arrested. There may be remissions also in the individual symptoms. The loss of self-knowledge is a very important symptom; for, although it is common for persons of unsound mind to fail to realise that they are unlike themselves and unlike others, this lack of self-knowledge is most marked among the general paralytics. The general paralytic seems incapable of knowing himself, and of recognising or comparing his past and present states. No amount of experience enables even a medical man to recognise the disease in himself, though he may indeed have been an asylum medical officer. Such a one may be hypochondriacal at the onset of the disease, and in dread of it; but, when he has once passed into the buoyant stage, he is quite incapable of realising his state, indeed will take a visitor round his wards and point out other patients who have similar symptoms to his own, without being able to recognise them in himself. This want of self-knowledge leads also to the grandiose ideas, the egotism, and even to the change of notions as to personality which partly depend on them. Patients may lose the feeling of self, and this may go farther, so that they have confused ideas of their own identity, and may even lose themselves altogether. I have met with patients who sought for themselves, and even took off their clothes to see if they were really there.

Megalomania.—Exaltation of ideas may depend on loss of thought-control, each idea, as it passes through the mind, being seized as real and appropriate. The mind is thus coloured by every passing wave of thought, and naturally assumes the happy guises of the great.

Exaltation, though common in general paralysis, is not invariable, nor is it confined to this disease. It may be present in ordinary and in alcoholic mania, in delusional insanity, and in paranoia; the exaltation of general paralysis differs, however, from the other forms in its universality and benevolence. The general paralytic at one moment is an actor, at another a bishop; he sees no inconsistency in being at once a god and a jockey, a poet and a millionaire; as a rule he is benevolent in his exaltation, being willing to grant power and wealth to those about him. The patient with monomania of grandeur as a part of delusional insanity is usually content with his one great idea, and is not in the least disposed to give up his power or to share it with others.

Exaltation is not only associated with the maniacal or expansive form of general paralysis, but may occur also in the other forms of mental disorder which may form part of it. I have met with melancholic general paralytics who have thought that millions of devils were torturing them; and I may refer again to more than one patient who supposed his bowels to be obstructed by millions of tons of fæces. In other cases delusions with ideas of persecution have been associated with notions that all the world was in league against them.

It is noteworthy that in general paralysis there may be a dual consciousness, so that while the patient talks of his millions of gold and diamonds he may yet remember the state of his balance at the bank, and act consistently therewith.

Micromania is a name used in contrast to megalomania and to indicate what Dr. Mickle calls "belittlement." This occurs more frequently in general paralysis than in any other form of mental disorder; but it is rare even in this. The patients say they feel so small they can go under doors, or they can get through cracks and keyholes. This feeling of belittlement may be associated with some cutaneous anæsthesia; as the opposite idea of greatness or swelling of body may depend on hyperæsthesia. Micromania is met with in certain other cases, but chiefly in senile degeneration, or mental degeneration of one kind or another. I have met with senile patients who spoke of themselves, and of all about them, as "little things."

Emotional instability is very rarely absent in general paralysis, and is often one of the earliest symptoms; there is a tendency to exaggerate both the joys and the sorrows of life; usually it is marked by increased irritability, so that the formerly docile, good-tempered man becomes hard to live with. In rare instances the change is of the opposite kind, the bad-tempered man becoming more easy to manage. Crying and passion are both common in general paralysis, and it is well to remember that hysteria occurring in a middle-aged man is very frequently an early symptom of general paralysis.

The general paralytic is pleased with a feather and tickled with a straw; he will sob over the penny novel and weep over the vulgarest drama. The ease with which men are affected by emotional causes is a gauge to their stability, and is a good indication of the decay produced by age or disease.

The defect of will-power, already described as one of the well-marked features of the disease, and the *facility* (of Clouston) are seen not only in emotional weakness but in the want of will which makes it often easy to guide these patients for their welfare; determined and obstinate perhaps if argued with, they may be diverted by changing the direction of their thoughts. Want of will may show itself in some cases as true *folie de doute*.

Memory is affected in very various ways: one of the earliest complaints made by patients themselves is that their memory is not what it was; but, as this statement is made by most men over forty-five, it does not attract much attention. Often the memory seems to be more seriously affected at first than later, probably because fewer calls are made upon it; the loss of memory is slow and progressive, in some cases the defects suddenly follow fits, which may leave a distinct gap. It begins with failure in the more recent and more isolated facts; then it leads to a strange jumble of ideas and a mixing up of the past with the present—the real with the ideal. The memory may seem to recover itself during remissions. Generally speaking, the loss of memory is not nearly so well marked as in alcoholic paralysis.

On the *sensory side* there is usually loss of acuity in one or more of the senses, but it is not common in general paralysis to come across cases of progressive failure ending in complete blindness or deafness; though I have often met with cases of more or less complete blindness, or deafness of one ear, which has proved transitory. There is no special change in the discs associated with the disease; in some cases there is an excess of vascularity in the early stages, in others atrophy; and in some the vessels seem to be placed in channels which have rather dark lines along their edges. Sometimes, towards the close of the disease, there is marked optic degeneration; at others temporary or permanent blindness will affect one or both eyes. It is probable that in the optic nerves, and in other peripheral nerves, degenerative changes generally take place if the patient live sufficiently long.

The pupils are generally unequal; the inequality varying, one pupil being larger one day, the other another. The pupils are frequently irregular in outline, but the most important point, in my opinion, is to ascertain whether they react to light and accommodation. We often meet with ataxic symptoms in which some reaction to light or accommodation is retained, though this is much less than normal; and there is little proportion between the spinal and the pupillary symptoms. It is common, however, to meet with small pupils in cases which began with locomotor ataxy; and it is also common to see dilated pupils in rapid cases, and in the later stages of the disease. There is no direct relation between the mental symptoms and the dilatation of the pupils; nor is there any direct connection between the dilatation of the pupil on (say) the left side and the exaltation of ideas. The pupils, then, are generally abnormal, they vary from time to time, they generally react defectively to both forms of stimulation, and they are often irregular and eccentric in form.

It has been taught in France that in general paralysis there is loss of power to detect pepper. I have tried to find this symptom but have failed, and I do not attribute any value to it. Many cases of hyperæsthesia have been described, and I have met with a few. In some there is only a local sensitiveness, which in most cases passes off; in other cases patches of anæsthesia occur, as I shall state presently, but as yet we can attach no special value to these symptoms.

Headache or head tenderness, neuralgia, and other nerve pains have been described by Batty Tuke and others; but it is not a common experience for me to meet with these.

Hallucinations and illusions of the various senses may be present at any time of the earlier stages of the disease. In my experience they are not so common as in other forms of mental disorder; but it is a mistake to think that they are rare. Dr. Mickle says that more than half his patients had hallucinations during one period or another. Those of sight and hearing were the most frequent, and were about equally common. Many general paralytics say they see and hear things, as the outcome of their boastful delusions; they think they are God's messengers, and must therefore hear from Him. There is nothing special in the character of the hallucinations, which may be single or multiple, pleasant or painful, constant or occasional. In some few of my own cases the hallucinations followed the same course as in systematised delusional insanity, and were associated with ideas of persecution, of spies or detectives, of whisperings and annoyance.

Common sensibility may be reduced or almost annihilated; I have known patients rub holes in their bodies or their limbs; and one case of a man who slept placidly while his hand was being roasted. It would seem that there may also be deficient reflex sensibility, so that in them there is an especial danger of choking by the food in the throat, which is not duly stimulated to swallow.

The *disorders of muscular or motor control* are many and characteristic; they are of two classes: the passive, as seen in defective reaction and defective expression; and the active, as seen in disorders of articulation, writing, and gesture. The face wears a mask, the cheeks are flabby without lines or folds; in some cases the patients look as if they had blown out their cheeks, in others the frontal muscles have to be fixed before the rest of the face can be moved. Besides loss of expression there is loss of control, as seen in the very tremulous over-action or ill-directed action. In many cases in which the knee-jerks are exaggerated there is also great excitability about the facial muscles; the tremor is usually most pronounced about the muscles of the lips, the tongue also being very greatly affected; for a time indeed it is ataxic, so that its movements are irregular and awkward. There may or may not be fibrillar tremor, but I do not think this is very common. The tongue from being ataxic becomes more paralysed and rests on the teeth, taking an impression from them along its sides. Certain words are not readily pronounced. I believe it will be found that some general paralytics fail in the labials, others in sibilants or dentals. Tremor of tongue and difficulty in speech are greatest after mental or moral excitement, and after eating. There is often loss of control over the cheeks and lips, so that saliva flows over the chin, or food collects about the gums.

Various forms of disorder are associated with the function of mastication; thus there is often a tendency to smack the lips, or to movements like sucking, grinding of teeth, or swallowing. The grinding of the teeth may go so far as to fracture them.

The *handwriting* in general paralysis is almost pathognomonic. In the earliest stage the patient often writes voluminously in a rather shaky hand, and it is noteworthy that the words are often left incomplete; syllables are omitted and the letters are not attached one to another. In some cases, however, the handwriting improves in legibility, for the patient, finding his inability to write quickly, begins to write large text like a child; yet even then the letters are often separate, and the words shortened. Although the writing has much the character of that of an old man, in this latter case the shakiness is not associated with the separation of the letters. In alcoholism, however, the shakiness and the elision or dropping of letters may occur together.

Muscular defect in gesture is seen in the orator and the actor, and may give way to ludicrous exaggeration of the natural expression. The gait varies greatly, there being many grades between the extreme ataxic and extreme spastic walk. In some cases the symptoms may vary, so that a patient who at one time seems hopelessly ataxic, at another may lose this weakness. In many cases the symptoms differ on the two sides. There is very often a great restlessness, which on the muscular side is seen in incessant movement or perpetual writing of letters.

Knee-jerks may be absent, normal, or exaggerated; or they may differ on the two sides: they may also vary from time to time.

Fits and Palsies.—During the progress of general paralysis fits occur commonly, but by no means universally. These may be the earliest symptom, though more frequently they occur in the later stages of the disease. The attacks have been called, according to their severity, congestive, epileptiform, or apoplectiform seizures.

I have already pointed out that the occurrence of hysterical fits in middle-aged men is of serious import; I need only add here that it is not uncommon to meet with cases in which hysteria was the first symptom of general paralysis. A hard-worked man who has had a great deal of worry and anxiety, when consulting the doctor, bursts into tears, sobs, then laughs, and for a moment cannot speak. Such emotional instability may precede the other symptoms by a year or more.

A slight attack of giddiness, some temporary loss of consciousness similar to that of petit mal, sudden transient loss of speech, blindness, or deafness may mark the onset of the disease; such symptoms pass off and recur. They may be present, under stress of mind or body, at irregular intervals several years before the other and more easily recognised symptoms of general paralysis are noticed.

Congestive or other seizures may start the active mental disorder: thus directly after a fit a man may show great mental excitement; but in the great majority of cases the fits first appear after the patient has passed into the second stage; so that some have spoken of this as the "fat, fatuous, and fitty stage." It is common experience that patients grow fat before the onset of the fits. There may be slight warnings, resembling attacks of petit mal, before severe apoplectiform fits occur; but this is by no means constant, as the first fit may be very severe, or even fatal. It is common to meet with some gastric disorder immediately before the fit, and increase of temperature is not an unusual warning of this danger. The fits are very irregular in their recurrence; and though for some time, affecting the same centres as epileptic attacks, they may present similar features, yet it is common to meet with fits which do not affect the same parts in their recurrence. The seizures may continue for days together, the patient remaining in a *status epilepticus.* Fits are most common toward the end of the disease, and are often the immediate cause of death. As a rule, marked mental degradation follows the onset of the fits, but in a few cases I have met with temporary improvement after epileptiform seizures.

Though the seizures may be most marked on the sensory, motor, or vaso-motor (or "organic") side, yet, as a rule, in complete fits all these parts are affected. I have seen several cases in which temporary loss of sight or of hearing, generally on one side, has preceded other and graver symptoms; in others local or general hyperæsthesia was present. On the motor side sudden loss of power in one hand is the most common failure, though occasionally speech may be embarrassed while consciousness remains perfect. I think very gravely of any temporary loss of power occurring in middle-aged active men.

It is not uncommon to meet with so-called "bilious attacks," which may have a vaso-motor origin.

The fits vary greatly in form and in degree. I have already spoken of the slight passing attacks which occur most frequently in the earlier stages.

Next in order are the epileptiform fits, which cannot by inspection be distinguished from true epilepsy. They are most commonly associated with complete loss of consciousness, though I have seen a patient convulsed while at the same time he was conscious, and was swearing at his disorderly limbs.

As in epilepsy, these fits frequently start in a definite spot and follow a definite line of development; but this is not always the case. The epileptiform fits may pass off rapidly, leaving the patient tired, but ignorant of what has happened; but, as a rule, the epileptiform fits of general paralysis leave more physical and mental weakness behind them than a similar nervous discharge in epilepsy would do.

While the result of an epileptiform seizure is more marked in general paralytics than is a similar fit in an ordinary epileptic, it will be seen, on the other hand, that the apoplectiform seizures in them are followed by less marked and less permanent disorder than is a true apoplectic fit.

I need not describe the character of the epileptiform fits themselves, as they start from the same centres as do those of true epilepsy, and generally run a similar course. In the apoplectiform fits we have usually one-sided convulsions, the fits often starting with a distinct deviation of the head and eyes to one side, and with rigidity and convulsive movements. These tonic and clonic convulsions may recur for hours or days together, as a result of external stimulation, or spontaneously. During or after the convulsive seizure there is most profuse sweating; and there may be involuntary actions of bowels and bladder. The convulsions may be so severe as to fracture the limbs or the teeth. There is frequently pallor at the onset, but this is replaced by congestion of the face and neck, which may pass into lividity. The pupils may be equal but are commonly dilated; there is complete insensitiveness of the conjunctiva.

The fits of this description may leave a patient comatose, or he may remain for hours in a very unstable state; so that the slightest external stimulus will lead to a fresh outburst of convulsions. As a rule, the hemiplegia which follows such apoplectiform seizures resembles in all details that of true apoplexy; thus with right-sided hemiplegia aphasia will probably occur. This hemiplegia, however, is of comparatively short duration, passing off much more quickly, as a rule, than that following a coarser brain-lesion. I have seen an apoplectiform seizure, leaving temporary paralysis, followed by a seizure of the opposite side, associated with recovery of power in the side first affected. After a severe apoplectiform fit there may be coma, followed by maniacal excitement; which, passing off, leaves the patient hemiplegic. In some patients such fits recur for many months or even for a few years; but, as a rule, frequent fits mean speedy exhaustion and death.

In all cases of fits the treatment must be purely expectant: bleeding or severe purgation is not to be thought of; chloral hydrate, in 20-grain doses, administered by enema, has seemed in some cases to shorten the fit and reduce the excitability. During the attack the patient should have his clothes loosened, and he should be kept from all sensory stimulation as much as possible. Unless very weak, it is well not to trouble about feeding by the mouth for a while, for the attempt will probably give rise to new convulsions. I do not think any good is to be derived from sinapisms or the like. In some of my cases amyl nitrite was given at onset of the fits, but I cannot report any favourable result from it. Under the head of apoplectic seizures some observers class the paralytic seizures, which may come on during general paralysis, in which a limb or part of the body may suddenly lose power for a time without any convulsions. These cases resemble apoplexies due to brain-softening.

The paralysis generally increases, and may become associated with contraction of the limbs, even to an extreme degree. The contraction may have a hemiplegic or a paraplegic distribution. With great amount of disablement muscular strength may be maintained. I have met with men who could register a very high power on the dynamometer, yet who could hardly direct a pen.

In some of the cases of general paralysis with ataxic symptoms I have met with various changes in the skin. In one case the joints were affected by Charcot's disease, and symmetrical bullæ formed about the feet; there was also a marked and persistent *cutis anserina* and an abnormal growth of hair on the legs. I know of no special odour associated with the disease. Herpetic eruptions may occur, more particularly in the earlier stages of the disease. Carbuncles may play an important part in general paralysis; they may usher in the disease or they may bring it to an end; again, I have seen very prolonged remissions follow serious carbuncles. There is nothing characteristic about the temperature of general paralysis; many observations have been taken, and these show that in some cases the disease is associated with a slight persistent rise in temperature, this rise being higher, as a rule, at night. During the middle and part of the later stages the temperature is often normal or subnormal; and I have known a chronic patient have a subnormal temperature for many months. Towards the end, when there are secondary complications, the temperature varies greatly and may be very important as a warning of such secondary disease—of pneumonia, for example. Before the onset of convulsions, and during and after these, a rapid and marked rise may occur. The rise is associated with or followed by profuse sweating.

General Nutrition.—Usually with the early stages of the disease there is a tendency to waste; later, to grow fat, although afterwards the fat is reabsorbed and wasting sets in again. There is still a great tendency to hæmatoma. Brittleness of bones and defective power of resistance are common, but at the same time the power of repair of injuries is often maintained at a high standard. I have no doubt that in some advanced cases marked arterial changes occur; but I cannot find any evidence of early or widespread arterio-capillary fibrosis: nor do I find grounds for believing that changes similar to those met with in Bright's disease are common. Yet I have often found evidence of syphilitic changes in the arteries of the brain. There is a liability to subcutaneous hæmorrhage; hæmatoma and pachymeningitis being among the results.

Capillary congestions are frequently seen over the malar bones; and in some of the hollow viscera—such as the bladder—they may lead to hæmorrhage. There is some change in the blood itself; but, notwithstanding the observations of foreign observers, I think nothing characteristic has been discovered in it. The skin becomes sallow and waxy, erythematous disorders occasionally appear, and if the nursing be inefficient bedsores will form. There is a great tendency to profuse sweating after fits, and this may lead to sudaminal rashes. "Tache cérébrale" does not seem to me to be of any value as a symptom, at any stage of the disease; it is common to many and various diseases, and may indeed be found in the normal state.

The *pulse* in general paralysis varies greatly: not infrequently in the earlier stage it is above the normal in tension and number; it soon loses power, often remains rapid, small and compressible, and may become very slow, when it points to increasing weakness of the heart. The pulse rises, as a rule, with the temperature. There is no characteristic sphygmographic tracing of general paralysis.

Respiration varies, as a rule, with the pulse. After fits it is common to meet with Cheyne-Stokes breathing, which may be of short duration or may persist; in a few cases respiration becomes very slow. Sleep in the first stage of general paralysis is often good, but the patient does not appear to need so much as usual; he will sleep profoundly, wake early, and begin his restless day betimes. In some cases this will lead the patient to the extravagant notion that he has discovered the root of all evil to be too long a rest in bed. Sleepiness is, however, no uncommon symptom of the earliest stage; patients may sleep almost as soon as they sit down; they sleep over a meal, and sleep well at night also. One teacher has gone so far as to assert that sleeping after meals is a cause of general paralysis; it is certainly a common symptom of it. In the second stage sleep is generally more normal; but towards the end there is restlessness, associated with the bodily discomfort of incontinence of urine and, in neglected cases, of bedsores.

The *urine* of the general paralytic presents no constant abnormal quality; I have met with sugar, and also with albumin, and not infrequently I have noted the high specific gravity, with abundance of lithates, which corresponds with the rapid tissue-changes of the body. Moreover urea and uric acid in excess, or sulphates in excess, are common. Occasionally, after very severe convulsions, I have met with albumin in the urine.

Remissions of the symptoms, both mental and physical, may occur in most of the periods of the disease. These are most common in the earlier stages, and they are more frequently met with in patients who have begun with symptoms of maniacal excitement. In part this is due to the fact that such patients have accentuated their symptoms by giving way to alcoholic and other excesses. The remissions may be complete or partial; thus, as I said, both mental and physical symptoms may disappear for a time, but it is much more common to meet with cases in which the mental symptoms subside while the physical ones are progressing.

I have met with one patient who, while excited, showed little or no ataxy in his gait, but who, as soon as his mind was more clear, was quite unable to stand alone; in this case there were several remissions, each associated with similar symptoms. I think that the symptoms which are most persistent are those affecting the finer adjustments, such as speech and writing. During the remissions the patients may resume their professional work for a time, and may rejoin their families; though, as a rule, the return to old ways leads to rapid recurrence of the symptoms. It is rare to meet with patients who have had more than one complete remission. Acute stages may be followed by intervals of quiescence, and these again may be followed by excitement; but after each period of excitement there is a marked degradation in the patient's state of mind. These cases with recurring attacks resemble in many ways the general paralysis of the double form.

Arrests, if not remissions, may occur even when the patient has passed into the paretic stage; and, in one case at least, I have seen the disease arrested for many years after the symptoms had gone so far as to lead me to think the patient was dying of fits in the last stage. Remissions may follow injuries or acute diseases, such as pneumonia: or they may follow suppuration, or some skin affection.

Terminations of general paralysis may be natural or accidental. The natural end depends on the progressive disease affecting the whole system; thus exhaustion and slow death by heart or lungs may occur; or the exhaustion may depend on inability to assimilate proper food: besides the weakness there may be diarrhœa, bedsores, and vesical catarrh. Implication of the bulb may lead to irregular breathing, and to defect of power in respiration; and this may cause clogging of the lungs, pneumonia, and death. Phthisis, or at any rate degenerative lung disease, may end the case. Whether the phthisis be more often fibroid or not I cannot say. The fits of one kind or another, which may come on at any stage, may cause death either directly by their frequency, or be associated with some brain lesion or blood lesion, such as hæmorrhage, which may lead to death. Such are the terminations which I have called natural.

The accidental terminations of general paralysis may come by fracture of ribs leading to pleurisy or pneumonia; and by fractures of other bones leading to other complications. Bruising may lead to hæmatoma, which may exhaust the strength, or give rise to blood poisoning or to abscesses. Local hæmorrhages may follow slight bruises on the surface, or be shed into the membranes of the brain. Exposure to heat or cold may cause death. Impaction of food in the air-passages, or in the œsophagus, or the inhalation of fluid during a fit, or as a result of paralysis about the throat, may cause immediate or more distant death; suffocation may occur during a fit, if the patient turn on his face. Suicide may be the result of such a delusion as that the patient can fly; or it may be the result of mental depression.

Pathological anatomy.—In considering the morbid appearances of the nervous system in general paralysis we note at the outset that there are none, so far as our present knowledge goes, which can be regarded as peculiar to this disease. In the very great majority of cases, however, which have been regarded clinically as general paralysis, the autopsy reveals a complex of morbid conditions which, whilst perhaps scarcely so suggestive as the group of symptoms which go to make up the clinical picture, is nevertheless sufficiently characteristic to bear out the diagnosis founded upon the latter. As regards the brain, it may be affirmed that certain morbid conditions are found there in a more pronounced form in general paralysis than in any other disease in which this organ is involved.

The naked-eye appearances of the cerebro-spinal axis, and its enveloping parts, with which we are familiar at the autopsy of a case of general paralysis in which the disease has lasted the ordinary period, are as follows :—The skull-cap (sawn through at a standard level) is commonly heavier than normal. In some 25 per cent of cases hyperostosis is noted. The diploe is obliterated or deficient in about three-fourths of the cases; the sutures are more or less obliterated, or covered with calcareous deposit, and exostoses occur. The dura mater exhibits changes in about one-half of the cases, is then thickened, congested, and adherent to the skull-cap, especially along the sagittal and coronal sutures and over the frontal bone. The Pacchionian bodies are unusually prominent, causing marked pits in the bone above them. Calcareous deposit occurs in the falx cerebri. The inner aspect of the dura shows rusty discoloration, or localised blood-clot, which may be surrounded by membrane; or cystic formations may be present ("arachnoid cyst"); or membrane of various degrees of consistence and extent may be stripped off, especially from the most prominent part of the convexity of one or both hemispheres, or the temporal fossæ.

The serous fluid in the subdural and subarachnoid spaces and in the ventricles is turbid and increased in amount, 4 to 6 ounces escaping. The arachnoid, where it bridges over sulci, and over the space between the crura cerebri and optic tracts, is thickened and milky, or opaque and swollen, watery or gelatinous; and the leptomeninges generally are thickened, œdematous, hyperæmic, and show milky streaks along the course of the main veins; these conditions being most marked over the convexity of the parietal and frontal lobes. The frontal lobes frequently adhere to each other, the adhesions being easily broken down. The pia, on stripping, is found to adhere to the cerebral cortex along the summits of the gyri, though for short distances only, and at this stage not strongly. Indeed, at the usual date of death there is frequently no adhesion, the leptomeninges stripping, on the contrary, with undue ease.

Adhesions are commonly present over the frontal and parietal lobes, at their convexity; and are especially noted over the central gyri and the bases of the frontal gyri. The occipital lobe does not usually present adhesions. After removal of the pia the cortex at the site of adhesion presents a worm-eaten appearance.

Summing up in respect to the meninges—there is evidence of diffuse, chronic meningitis in over 80 per cent of the cases.

Of the brain there is a general atrophy: the gyri are shrunken, the sulci and lateral ventricles are widened, and there is a loss of weight (1250-1270 grams would represent the weight commonly found). The organ is flaccid; it collapses when placed upon a table, the hemispheres separating posteriorly, and the posterior extremity of the corpus callosum becoming torn—an indication of softening. Atrophy, though general, is especially noted at certain parts, such as the central and precentral gyri, the basal ganglia, the pons, and medulla. Softening is also general, with foci of special intensity; it occurs irregularly in the gray and white matter, especially in the parietal, central, and temporal gyri, in the island of Reil, in the external and internal capsule, and the lenticular nucleus; it is also met with in the basal ganglia. The focal softening is ascribed to blockage of minute arteries. There is often considerable atheroma of the main arterial trunks, and hæmorrhagic foci exist, especially in the centrum ovale, the basal ganglia, and the pons and medulla; these are ascribed to degeneration of the walls of the arterioles in conjunction with increase of the arterial blood-pressure. Frequently also there are traces of former hæmorrhages, in the form of cysts and scars. The cerebellum participates in the above degenerative changes, but not in a marked degree: here softening—general and local—and hyperæmia are the most marked naked-eye alterations.

The cortex cerebri exhibits foci of softening, general hyperæmia, or patchy discoloration. It is also atrophied, especially in the frontal and parietal regions. In the white matter similar conditions exist; and the perivascular spaces are dilated. The ventricles are dilated, and the ependyma of the fourth ventricle is congested, thickened, softened, sometimes gelatinous, sometimes tough; its surface is granular in varying degree. The same conditions of ependyma are found in a lesser degree in the other ventricles.

The cranial nerve-roots show gray degeneration and atrophy: these conditions have been noted in the olfactory bulbs and tracts, the optic tracts, the third, fifth, sixth, and seventh nerves.

In contradistinction to some of the conditions noted above we find exceptional states, chiefly affecting the coverings of the brain: of such is atrophy of the skull-cap, which is therefore lighter than normal; the dura mater may appear quite normal—this is less unusual than might at first be supposed,—or there may be evidence of pachymeningitis externa, and rusty deposits may be present on the outer aspect of the dura; granulations occasionally occur on the outer aspect of the arachnoid, and adhesions between dura and arachnoid. Adhesions between the pia and cortex may be very rare, or unusually situated, or absent; even though the case be not an advanced one. The ependyma may appear normal, or at most slightly granular; in about 20 per cent of the cases naked-eye evidence of disease in this structure is but slight.

Turning to the spinal cord and its investing membranes: there is naked-eye evidence of disease in the membranes in about 39 per cent of the cases. Adhesions exist between the vertebræ and dura mater; and the cord-tunics are thickened, opaque and hyperæmic, chiefly posteriorly. There may be blood-extravasation upon the inner aspect of the dura; but deposits, whether hæmorrhagic or calcareous, are much rarer upon the cord-membranes than upon those of the brain. Occasionally there are adhesions between the dura and the pia. The cord itself is chiefly softened, but sometimes indurated. The different columns, chiefly the lateral and posterior, may show gray degeneration; there is evidence in some cases of disseminate sclerosis. Lesions of the columns, manifest to the naked eye, are less commonly observed in general paralysis than in the ordinary degenerations of these parts. In some cases the entire cord is diminished in size; or there is diffuse softening. The cord-changes, as will be seen when histological conditions are referred to, are very various.

In acute cases, and in those which succumb in the earlier stages of the disorder, the naked-eye appearances, as might be expected, are less indicative of disintegration and degeneration; in place of these are exhibited the signs of acute disease:—the blood-vessels are everywhere congested, whether in the diploe, dura mater, leptomeninges, or brain-substance; flakes of lymph occur in the cerebro-spinal fluid, and there may even be, at points, pus-formation—purulent meningitis (Kaes). The membranes are tense, and on cutting them the full-volumed vascular brain projects, and its diminished consistence is noted. Adhesions between the pia and cortex are strong and extensive. Such adhesions are present in at least 70 per cent of autopsies of general paralysis. There may be recent blood-deposit in the subdural space.

Histological changes.—The Brain.—Seeing that when the disease has lasted its customary period, end-products only, of no particular importance, are met with in the brain, cases are selected, for the purpose of histological examination, in which death had taken place in an earlier stage; preferably those in which the duration of the disease has not exceeded one year. In such a case we find in the cerebral pia mater that the vessels are prominent, with thickened walls and abundance of nuclei. There is increase of the cell-nuclei of the sheath of the vessels which pass from pia to cortex, with thickening of the sheath. The septa passing from pia to cortex are thickened and increased. There is free exudation into the meshes of the pia. The trabeculæ between arachnoid and pia, as seen in a sulcus, are bathed in inflammatory exudate. The various structures of the cerebral cortex give indications of disease. Thus the blood-vessels are exceedingly numerous, distended with corpuscles, and tortuous; at points along their course they exhibit dilatations, at which points the lumen, on cross-section, is found occluded by a yellow mass, which takes aniline blue stains very deeply. The nuclei of the vessel-wall are greatly increased, and extravasated leucocytes are apparent. The coats of the vessels are thickened, and there is proliferation of the endothelial nuclei. Hyaline degeneration, or fatty change, of the vessel-wall may be present. Hæmatoidine crystals about the vessels point to extravasated blood. In connection with the increase of nuclei about the vessels and in the neuroglia it is to be observed that the shrinkage and disappearance of the tissues may be held to account for some of the apparent increase. The vascular lymph-spaces, subadventitial and perivascular, show collections of lymph-corpuscles and blockage. The perivascular sheaths become much distorted by the contraction of the vascular processes of the spider-cells.

The supporting structure (neuroglia) exhibits deep staining in many parts of a section; this is especially marked in preparations by the fresh method of Bevan Lewis. Instead of a ground-substance scarcely stained, there is found a mottled, patchy-blue staining between the nerve-cells, a granular or fibrillar condition. In other parts the staining is defective. With these conditions deeply-stained round bodies, apparently nuclei, are found in abundance; and glia-cells ("spider," "lymph-connective") are numerous and prominent, with large, deeply-stained bodies and numerous processes.

These spider-cells are especially noteworthy in the outermost layer of the cortex, in contiguity with the apical processes of the pyramidal nerve-cells, where their increase is earliest noted; and also in connection with the vascular sheaths. A strong "vascular" process passes from the cell to the vessel-wall, ending there in an enlarged extremity, a nucleated mass of protoplasm; and from the body of the cell other processes radiate. But these cells form also prominent objects between and around the nerve-cells; and it is noteworthy that their processes may be traced to the degenerate bodies and processes of the nerve-cells, and are found surrounding the latter. These conditions are best shown by the fresh method above mentioned. At a later stage the spider-cells are transformed into a fibrillar meshwork.

The nerve-cells and their processes exhibit degeneration: the latter are stunted, granular, and blurred in outline; later they are thin, with broken or irregular course, disintegrated, or destroyed. The apical process suffers early, a point to be noticed in connection with the great development of spider-cells in the outermost layer. The cell-bodies exhibit various changes; their contour is blurred, they stain irregularly, and show granules or fuscous change (formation of brownish - yellow pigment), sclerosis, and occasionally vacuolation. The nucleus is no longer distinct. The cell-shape is altered: it is swollen, irregular, locally defective; later it is atrophied, or merely a broken-down residue of pigment. There is, in the result, considerable destruction of the cells. In the smaller cells the nuclei appear swollen. The cell-nuclei in many cases show altered staining reactions, the reasons for which are obscure.

The nerve-fibres of the cortex also show changes; alike in the outermost layer (parallel to the surface) and in the intracortical radiations there is degeneration (wasting, disappearance) of the fine medullated fibres Tuczek). Some observers assert that this is especially the case in the frontal lobes, and in the next place is most frequent in the parietal and temporal lobes, and the paracentral gyri.

Ljubimow has described the degeneration, atrophy, and—in the later stages—disappearance of the "association-fibres" of the cortex: this degeneration proceeding from before backwards over the cortex. This nerve-fibre degeneration is regarded as a primary degeneration, independent of any inflammatory process. Colloid bodies are seen, especially in the outermost layer, which are ascribed to degeneration of the nerve-fibres.

In summing up the above histological changes, it may be said that they consist in atrophy of the nerve-elements, and hypertrophy and hyperplasia of the connective tissue.

Special attention has been drawn of late to the various changes which indicate a choking of the lymph-channels of the cortex, whereby lymph-stasis is induced. These are—hyperæmia (dilatation) of blood-vessels, with increase of their nuclei, thickening of their walls, and accumulation of protoplasmic heaps in the subadventitial and perivascular spaces; the overgrowth of spider- (secondary glia-) cells, with distortion of vessel-sheaths produced thereby; adhesions between pia and cortex, causing occlusion of parts of the epicerebral space, and of the communications therewith of the perivascular lymph-spaces.

Changes similar in character to those existing in the cortex are found in the area immediately beneath the cortex, in the medullary substance, the basal ganglia, corpora quadrigemina, cerebellum, pons, and medulla; but these are less marked, and have not claimed the attention which the condition of the cortex has naturally received.

Degenerative changes are present in the nuclei and roots of the cranial nerves (the vagi particularly, which helps to explain cardiac and pulmonary disorders), with evidence of vascular implication (pronounced hyperæmia, with thickening of the walls of the vessels).

The histological *changes in the spinal cord and peripheral nerves* are next to be considered. Changes occur in great variety in the cord, and are found in the majority of cases. Out of 145 cases Fürstner failed to find morbid alteration in 16 only. The tunics show chronic inflammatory changes; more especially there is evidence of chronic leptomeningitis; the pial septa are involved in this. In the cord itself there may be a diffuse degeneration, or irregularly occurring patches of degeneration (softening, sclerosis); more commonly the latter, in combination with changes in the columns. Degeneration is usually found in combination in the posterior and lateral columns, and most notably in the former; but sometimes singly, and then most frequently in the posterior columns. Fürstner finds that one side is regularly more affected than the other. The lateral columns are especially affected in the dorso-lumbar region; in the posterior columns degeneration is often seen along the whole length of the column, with degrees of local intensity. The anterior columns are but rarely affected, and never without disease in the posterior and lateral columns. The degeneration may be more intense in certain streaks; as in the posterior root-zones, or in Goll's tracts, or in the lateral pyramidal tracts. These degenerative changes consist in hyperæmia; the vessels being engorged, their walls thickened, and the nuclei of the latter increased. These vascular changes are especially prominent in the posterior columns. The connective tissue is increased, with nuclear proliferation, and enlargement and proliferation of the spider-cells; especially along the vessels. The medullated nerve-fibres show loss or swelling of myeline, the latter taking up the stain; or the myeline is granular, the axis-cylinders being interrupted or distorted. The cornua also exhibit changes in varying degree; there is atrophy and degeneration of nerve-cells, the latter being granular, swollen, translucent, with processes shortened (atrophied). The spinal nerve-roots (posterior and anterior) are also degenerate; their connective tissue is hypertrophied, from chronic inflammatory change; the nerve-fibres are atrophied. The lumbar and sacral nerve-roots are believed to show the most marked change.

The peripheral nerves show, in varying degree, parenchymatous degeneration (degeneration of the medullary sheath; swelling and atrophy of the axis-cylinder) and atrophy; and overgrowth of the connective tissue. The degenerative changes are more marked in the nerves of the lower than of the upper extremities.

The ganglia of the sympathetic system have been found to exhibit changes indicative of degeneration of the nerve-cells and chronic inflammation of the connective tissue.

Degenerative changes are also found in the muscles, including the heart and diaphragm (Mott, Campbell). There is fatty degeneration with disappearance of the muscle-fibre and increase of connective tissue.

Conditions indicative of degeneration have been described (Klippel, Mickle, and others) in various viscera, as in the lung, stomach, intestine, liver, spleen, kidneys. Concerning the nature and origin of these changes (whether primary or secondary to nerve-degeneration) it is not possible at present to give precise information.

Pathogeny.—The disease is one involving the whole nervous system; though the cerebrum, and more particularly the cerebral cortex with the investing pia arachnoid, is in the vast majority of cases the earliest seat of disease, and the locality in which its effects are most pronounced. The morbid process is usually first manifest and most active in the frontal and parietal areas. In some cases the encephalon alone shows indications of disease. Occasionally, to judge by symptoms, the cord or bulb would appear to be attacked first, and the brain later. As regards structures outside the nervous system, it may well be that these are attacked independently of this system, and not merely involved secondarily. Thus the fatty changes which occur in various muscles cannot be summarily ascribed to degeneration in the nerves passing to them; on the contrary, examination of these nerves in some cases has failed to show changes which would account for degeneration in the muscles (Mott). It is not possible at present to affirm how far the various degenerative states of other tissues are secondary and due to nerve-degeneration (impaired nutrition), or to vaso-motor disturbance, dependent on degeneration of the central nerve-tissues, especially the cortex cerebri; or are primary and independent, ascribable—like the muscular degeneration—to the influence of a toxin.

The nature of the pathological process remains obscure. It has long been, and still is, a subject of debate whether the primary change be inflammatory, affecting the vessels and interstitial tissue—a meningo-encephalitis, or a parenchymatous degeneration—the specific tissue, the nerve-elements, being primarily attacked. According to the first view the degeneration and atrophy which the nerve-cells and fibres exhibit are due to pressure from increase in the surrounding connective tissue; the second view is that the inflammatory changes manifested by the supporting framework of the nerve-elements are merely secondary in nature, "the result of decay of the neurons, and the irritation of the products of their degeneration" (Mott). It will not be possible here to discuss these debatable propositions; it may be affirmed, however, that the view that the primary change occurs in the specific tissue, the nerve-elements, is at the present time receiving an increased measure of support. In favour of it is the fact that the frontal lobe—the highest level centre—is the part earliest affected, there being no apparent reason why, were the change primarily inflammatory, the temporal and occipital lobes should not be equally attacked; yet these parts exhibit lesions to a comparatively slight extent. Further, Zacher, investigating the brain in an early stage (fourth to eighth week) of the disease, found a high degree of atrophy of the nerve-fibres of the cortex, with very slight vascular lesion. Others (Colella, Awtowkratow) have described alterations in the processes of the cerebral cells, and in the cells of the nuclei in the bulb, where the blood-vessels have been simply dilated. It has to be remarked, however, that, whereas vascular and nerve lesions are commonly mixed without a clear preponderance of one morbid condition over the other, in some cases the former, in others the latter are the more evident; in other words, the process would appear to be sometimes mainly inflammatory, sometimes mainly degenerative.

At the present time attention is being directed to the inquiry whether certain of the lesions of general paralysis can be referred to the action of toxic substances. Mott has suggested that the lesions found in various muscles are ascribable to toxic influence; and the latter may also be responsible for morbid states of the cortex cerebri which underlie certain clinical manifestations, especially the convulsive seizures. Varieties of histological lesion and of clinical state would be ascribable to variations in the nature and method of operation of the poison, and in vulnerability of tissue in individual cases. Such toxin may enter from without, or may be elaborated within the system (auto-intoxication).

These considerations would throw light more especially upon the origin of certain complications of the disease. But in the cases of general paralysis in which a history of syphilis has been clearly made out, it is legitimate to suppose the introduction of a toxin which has impaired the vitality of the nerve-elements, and which is fundamentally responsible for the disease itself, by producing a vulnerability of nerve-tissue which allows the various determining factors to become operative.

The interference with the lymph flow, which occurs in the course of the morbid process, and which results from occlusion of the lymph-channels, as indicated by the lesions above described, must have a deleterious effect upon the nerve-elements, and hasten their degeneration. The morbid development of the spider-cell—one of the conditions instrumental in bringing about this occlusion—is a feature to which much importance has been attached by Bevan Lewis, by whom this cell is regarded as the distal extension of the lymphatic system. According to this writer, the spider-cells in general paralysis, and in other diseased conditions of the nervous system, act as phagocytes or "scavengers," multiplying upon and removing the degenerate nerve-elements; they are also destructive of the living tissue. The nerve-elements are replaced by fibrillar connective tissue. This replacement is sufficiently shown in sections. It is ordinarily held that the nerve-elements are destroyed by the compressing action of the newly formed sclerous tissue. But in the hypothesis to which reference has just been made the replacement is regarded as a genuine degradation, the sclerous tissue being formed out of the effete material afforded by the degenerate nerve-tissue.

Further research is, however, needed ere a critical opinion can be expressed upon these views.

EDWIN GOODALL.

Diagnosis.—It is not always possible to distinguish between general paralysis and some other forms of mental disorder; each stage of the disorder has its difficulties. No single symptom can be considered as pathognomonic; to establish the existence of the disease we must prove the presence of both bodily and mental symptoms, which on the whole are progressive. In forming a judgment it is well to remember that there is no form of insanity which may not be associated with general paralysis. In examining male patients of middle age the question of general paralysis must always be taken into consideration. General paralysis is rarely detected in its earliest stages, which stages are not recognised until the disease is fully established. Neurasthenia may be the starting-point of general paralysis; therefore in every case of extreme nervous weakness it is well to examine the state of the pupils and the knee-jerks, and to look for any evidence of the emotional instability and change in temper which are common in this disease. If hysteria occur in middle-aged men it is well to look out for the above signs of degeneraion; and in both neurasthenia and hysteria changes in articulation and n writing must be closely observed.

Convulsive or paralytic seizures of a transient kind must not be overooked, as they often precede the more marked signs of general paralysis. The occurrence of such fits or palsies for the first time in men without ardiac or renal disorder, the rapid passing off of the symptoms, and the resence of some such physical signs as pupillary inequality, or sluggishess with defects of expression, suffice to clear up any doubt between pilepsy and general paralysis. The perpetration of criminal acts by men reviously moral is often found associated with early general paralysis; ut in a court of law some marked symptom such as fits, pupillary hanges, or well-marked change in character of a progressive type not ttributable to alcoholic excess, must be relied upon. If simple depression or slight hypochondriasis suggest early general paralysis, physical signs of degeneration must be looked for; and I think the most important thing is to observe whether there is any inconsistency between the mental complaints and the physical states; thus the hypochondriacal general paralytic complains, but still gains flesh and appears well.

The diagnosis in the acute stage rests chiefly between mania of various kinds and the maniacal onset of general paralysis. I do not think it is possible to make a distinction between acute delirious mania which may end in general paralysis and acute delirious mania which may pass off, or may end in death.

In all cases of delirious mania not due to alcohol, in which there is a history of syphilis, it is well to be on the look-out for general paralysis. I think in the latter there will probably be found more marked alteration in the articulation, and more marked tremor about the muscles of expression; but in acute delirium also these may be present to some extent. Pupillary inequality may aid diagnosis, but in either case the pupils may be dilated and rather immobile. In ordinary acute mania there may be great difficulty and delay in coming to a definite decision; and the difficulty is the greater when the mania depends on alcohol. As a rule in mania the sleeplessness is better marked, and there is rather more persistence in one line of thought than in the acutely maniacal stage of the general paralytic, in whom there is greater mobility and more rapid change of ideas. In general paralysis, again, the exaltation is more marked and more variable than is common in mania. In general paralysis drugs and alcohol seem to have a greater effect than in maniacal cases. If in mania we meet with great variability, intolerance of drugs, pupillary inequality, and change in speech and expression, general paralysis may be suspected. If the earlier symptoms be melancholic there is nothing characteristic in these alone. The melancholia may be of any form; but if with melancholic symptoms there is "physical inconsistency," that is, if the patient gain flesh, though still complaining that he is ruined, or is a miserable sinner, and if it be found that his pupils are unequal and indolent, and his speech defective, then general paralysis is to be feared. The occurrence of convulsive seizures often clears up the diagnosis. In some cases when stupor occurs early in general paralysis great doubt may arise: in these cases, if the patient gain in flesh without losing the apparent melancholy, we should search for inequality of pupils and for tremor about the facial muscles; but, again, it is common for the nature of the disease to remain unsuspected till convulsive seizures occur, or till the patient passes into a state of excitement. It is noteworthy that the majority of melancholic general paralytics have distinctly hypochondriacal ideas—the notion that their bowels are occluded being a common one.

In progressive dementia there may be great difficulty, for the mental symptoms are exactly the same in this and in general paralysis; but, as a rule, the progress is more rapid in the latter than in ordinary dementia: the latter, again, is more often associated with age or some definite cause of physical weakness, while general paralysis occurs in middle life. If in progressive dementia there are signs of muscular weakness—such as tremors and pupillary inequality and defective articulation, general paralysis must be suspected. The cases of simple general paralysis seen in general hospitals are usually of this type; convulsive seizures frequently reveal their true nature.

Alcoholic disorders and general paralysis.—As already pointed out, the symptoms of general paralysis are also the symptoms of alcoholism; hence, unless the history be very clear, it is impossible to form a correct opinion at once. In some cases, indeed, intemperance and increased nervous instability are symptoms of general paralysis. The excitement of delirium tremens resembles the delirious onset of general paralysis; and it is well to remember that delirium occurring in a man as the result of comparatively slight alcoholic excess may depend on this kind of degeneration. The acute alcoholic patient, as a rule, sleeps worse than the general paralytic, and has much more marked visual hallucinations of the terrifying kind. The diagnosis must be made from the history, and the existence of some change in the pupils, speech, or writing; but in spite of the most careful consideration mistakes will occur, and time alone can determine the nature of the disease. I think that in every acute case of mental disorder in which there is any suspicion of alcoholic causation time for observation must be insisted on. Convulsive seizures may result from alcoholic excess, and I do not think anything but time can decide on the cause of these fits. Chronic alcoholism resembles the second stage of general paralysis in many ways; but as a rule there is more evident loss of recent memory in the alcoholic than in the general paralytic; and the drinker "makes believe" more, talking of what he has been doing and seeing, when he has in fact never left his room. There is no pupillary change, the reflexes may be normal, and, though the facial expression is changed, there are not the tremor and the defective articulation which are so common in general paralysis.

Lead-poisoning will produce any of the ordinary symptoms of general paralysis, and may, indeed, be the cause of the disease; we must make sure, therefore, from the history and the physical signs that lead is the cause. The blue line on the gums, the tenderness, and the early paresis of the extensor muscles of the arms are the chief points of distinction. I have never seen a case in which morphia or any other vegetable alkaloid has produced symptoms which could be confused with general paralysis; but I have seen several chronic takers of chloral, with loss of mental and muscular power, who were not easily distinguished at first from general paralytics. In these, as in drunkards, removal of the drug and careful observation are our only safeguards. Influenza has in many cases set up degenerative changes which turned out to be general paralysis; it is therefore not to be forgotten that general paralysis may follow rapidly on influenza.

The *convulsions of kidney disease* may be mistaken for general paralysis; and, in some cases of alcoholic kidney disease, dementia may have been slowly coming on before the fits, and difficulty may thus arise; but, as I have said, albuminuria is very rarely met with in general paralysis.

Syphilis and General paralysis.—As I have already said, syphilis is a common cause of general paralysis, and the affinities between some cases of brain syphilis and general paralysis are very well known. The chief point is this, that in syphilitic brain disease apart from general paralysis we expect to do good by specific treatment, whereas in general paralysis this treatment fails; the nocturnal headache and oculo-motor palsies are very common in brain syphilis, and very rare in general paralysis. In cases of progressive mental decay, in which there has been syphilis with symptoms pointing to coarse intracranial lesion, the implication of any cranial nerve is against general paralysis; though it must be remembered that in general paralysis such a nerve implication may occur, especially in the prodromal stages. That a widespread syphilitic cerebral arteritis is often mistaken for general paralysis of the insane has been pointed out by various observers. Locomotor ataxy may be due directly to syphilis or be a part of the general paralysis.

I do not think it necessary to describe the so-called *pseudo-general paralysis*. Suffice it to say that in many cases of syphilis there are brain symptoms which prove not to be progressive, and which are not part of a steady process of decay.

Tumours of the brain, whether cancerous, syphilitic, or gliomatous, may lead to difficulties in diagnosis. A history of cancerous growth elsewhere might assist the diagnosis; but the presence of local headache, of vomiting, of optic neuritis are more important distinctions. The better-marked effects left after convulsions due to brain tumour are likewise important. The mental state of the patient with brain tumour is, as a rule, one of apathy rather than of excitement or disorder.

Tumours about the pons or cerebellum are also at times difficult to distinguish from general paralysis; but, as a rule, the staggering gait is greater than with general paralysis of the same duration; moreover optic neuritis is pretty sure to be present, with vomiting and general affection of certain groups of neck muscles.

Disseminated sclerosis may in some cases lead to confusion; when this disease was first demonstrated in England, by Dr. Moxon, I saw many cases of general paralysis, in general hospitals, which were regarded as insular sclerosis. Insular sclerosis usually occurs in young persons, and its progress is slower; there is a difference in the speech which is more staccato, the movements are rather jerky than tremulous, at least for a time; there is often nystagmus, which is very rare in general paralysis, and muscles are often picked out in various groups; the special degradation does not follow the lines of latest and most special development.

Paralysis agitans may be associated with mental weakness, but, generally speaking, the character of the movement is quite unlike that met with in general paralysis; the age of the patient also aids the diagnosis.

Sunstroke may give rise to nearly all the symptoms of general paralysis; and a history of sunstroke occurring in a tropical climate and apart from alcoholic excess must make one hesitate in giving a final judgment on a case.

Epilepsy is hard to distinguish from general paralysis. If the fits alone are considered, there is nothing distinctive about them; they may affect the same centres in the same way: but whereas epileptic fits usually pass in a short time leaving no paralysis, in general paralysis the parts affected are almost always paralysed or enfeebled for some hours or even for days after a fit. The epileptic fit, if the first, does not often leave much mental deterioration, while in general paralysis a fit often leaves the patient docile and quiet, however excited and grandiose he was before it. In some cases slight attacks, as of petit mal, occur in general paralysis; and in such cases the difficulty is the greater because there is but little mental change after or with them; in such cases you must depend on the pupillary or other general symptoms of general paralysis.

Apoplectic fits occur in general paralysis, and, as with epilepsy, they cannot at first be distinguished from true apoplexy; as a rule, however, they pass off in the course of a day or two—thus proving that, though implicating one-half of the brain and body, the lesion was rather functional than organic. The frequency of fits of this kind may also indicate their nature. I have known a patient have a dozen severe apoplectic fits, yet recover a fair amount of mental and physical power. In some cases apoplexy proper may appear to be the starting-point of the general paralysis.

Probably the greatest difficulty arises in the cases of men who have many of the symptoms, both bodily and mental, of general paralysis, but who prove later to be suffering from mental and moral perversion, and not from progressive disease. I can best make this clear by describing a case.

A middle-aged man of very neurotic family, married, with no family, with no history of syphilis, who had led a moral, respectable life, but who had suffered from various gouty symptoms, had a severe bodily illness from which he slowly recovered. After this he was a changed man, his conversation was no longer refined, he spoke openly of sexual matters, and made ribald jests before his wife and family. He then got entangled with a notoriously profligate woman, and was seen with her openly. He even proposed to introduce her to his wife and friends. He could not see the impropriety of his conduct, but suggested that his wife should follow similar ways. He retained his affection for wife and family, although still leading this most immoral life.

This state of things went on for many years, the man saying that now at last he had learnt how to enjoy life. In such a case I have seen pupillary inequality, unilateral sweating and some hesitation in speech; but no defect in gait or expression, no loss of memory, and no progressive decay.

I can only repeat that although such cases are almost always considered to be general paralysis, yet the patients may go on unchanged for many years. I have heard them described as cases of satyriasis.

Criminality or General paralysis.—First, as an early symptom of the disease, the patient may lose control and be guilty of impulsive criminal acts; in a moment he may commit a murder or cause grievous bodily harm; he may destroy property or do other acts which are simply due to his defective power of control; like many persons who have suffered from injury to the head, he may be more easily affected by alcoholic and similar stimulants.

Suicide may result from similar want of control.

Wrongful desire of one kind and another may arise, to be gratified without regard to social law; thus the patient may steal, though in my experience the general paralytic who commits theft does so from a conviction of his great wealth, and his right to all he may desire.

To complete this part of my subject I will now repeat one or two points already referred to.

Male hysteria may simulate general paralysis, yet general paralysis may begin as hysteria. Exaltation of ideas may occur in mania, especially if due to alcoholic exaltation; it may be the natural result of defective control in youth—an overgrowth of conceit, or it may result from delusions with an apprehension of being noticed or watched; it may be a steady growth of the imagination, the "may be" becoming a reality; but in general paralysis, as a rule, the exaltation is general and of a benevolent character.

It is difficult to tell whether a patient be suffering from mania of the double form, or from general paralysis of the double form; and time alone can settle the question.

Paraplegia in its various forms may be associated with general paralysis, and set in before, during, or after the mental symptoms; therefore in every such case the mental as well as the bodily weakness must be studied.

Sexual faults or crimes are commonly met with; these acts may arise from exaggerated or inflamed desire, or from loss of control. Indecent exposure may arise from unnatural desire or from simple forgetfulness. Rape and assault on children, indecent and criminal assaults, are apt to occur likewise. In the earlier stages of the disease there is almost always great increase of sexual desire, and some increase of sexual power may be present; and this may last for a considerable time.

The period when criminal acts are most likely to be done is before the full development of the more marked symptoms, or in the first period of their manifestation; hence it is of great importance that careful examination should be made for physical signs of degeneration in suspected individuals.

As far as *mental capacity* is concerned, it is certain that some general paralytics become unfit to dispose of their property long before they can be certified as insane; for in the earlier stages of the disease great alteration of temperament and disposition takes place as well as a tendency to act without due judgment; and later the memory may be defective also. Thus, as the result of progressive brain disease, a patient may be alienated from his relations, attracted by some designing person, probably a woman, and, as the result of these perverted feelings, may make an unjust will. On the other hand, a general paralytic can make a perfectly reasonable will, even in the late stages of the disease, provided the symptoms chiefly affect the muscular side of him. It was even allowed in the case of Crabtree, tried in 1894, that a general paralytic during a period of remission may make a valid will, though he had been previously in a state of apparent dementia, and had had fits.

A general paralytic, indeed, may be guilty of crimes of various kinds as a result of his disease, but may be held capable, nevertheless, of making a will even at an advanced stage of the disease (see Brown *v.* Penn, Nov. 1895).

Prognosis.—This is rather concerned with the probable course and duration of the case than with the result; I can claim very few recoveries from undoubted general paralysis. As I have said, sometimes early threatenings of the disease, occurring in likely subjects, may pass off. In some cases remissions may be so complete for many years that recovery is alleged; but the end of nearly all these cases proves that the respite was but arrest or remission. I will not say that no case of general paralysis ever ends in recovery, but it is very rare. The more acute the onset, the greater the prospect of remission; and the stronger the neurotic heredity, the greater, I believe, is the prospect of remission, and of the disease running an irregular course. It is very rare for more than one complete remission to occur in any case, save in those of the double form. In a few cases the quiet demented stage may last for years. General paralytics live longer in asylums than in their homes; they live longer in small asylums than in larger ones, and they live longer in the country than in towns. It is not possible to say whether a particular case will run a short course or a long one; yet as a rule the higher the temperature the greater the danger of rapid decline. The presence of fits generally hastens the progress; but in rare cases fits seem to arrest the disease for a time, or even to lead to remissions. Though a patient may have been quiet and placid in the earlier stages it is not certain that he will not have a maniacal outbreak later. The cases of general paralysis of the double form, or of the circular form, generally last longer than others, their duration being extended by the restful periods and prolonged remissions. A clear history of syphilitic infection within five years will give more prospect of amelioration; but, however distinct the history of syphilis, if of many years' standing I do not think it really affects the prognosis; that is to say, if the diagnosis of general paralysis be confident.

Treatment may be divided into general and special. The first consideration is whether the patient can be properly treated at his own home, or at any rate out of an asylum. Seeing that these cases are almost hopeless, it is well in all cases to decide at once in favour of removal from home to definite asylum care, except in those of simple progressive dementia without excitement, very distinct depression, or marked delusion. The treatment of a very wealthy general paralytic may be undertaken in a private house with large grounds away from a city; but even then, in my belief, it is neither the best nor the kindest treatment. Whether away from home or not, the patient's nearest relations, more particularly his wife, should not live with him. The less emotion there is in the life of the general paralytic the longer will his life last. Indulgence of the sexual passion is injurious, and the prospect of children begotten by such a father is gloomy. The wife in such cases, if installed as nurse, will probably break down in health; and I frequently see neurasthenia as the result of wifely devotion to general paralytic husbands. Sea voyages, though so often recommended,—only it seems to me in order to shirk the alternative of an asylum,—are rarely, I think, of any service.

The golden rule of treatment is complete rest. The simplest country life, with as much sleep as the patient can take, is to be desired; only moderate exercise must be allowed, and of course riding and cycling have to be given up. During periods of remission it is difficult to prevent the return of the patient to his own home; but this return is almost always followed by relapse. It is better to send the patient away for the interval; possibly for a short sea voyage, or else to the house of some medical man living in the inland country. The diet of the general paralytic must vary with the stage of the disease; in all I think alcohol is to be avoided. The simpler the food the better; fish, fruit, and plain food is better than much meat and fancy dishes. If any stimulant be found necessary it should be as weak spirit and water or light wine. Tobacco may be of service in the earlier stages of restless excitement; but there is a danger that the patient may smoke too much.

The general paralytic must be warmly clad, and his clothes carefully looked after, as he is very careless of himself.

I do not think that electricity in any form is of service. Specific medical treatment has made but little progress as yet. If antisyphilitic treatment has not been fully tried, I recommend, if the patient be quiet enough, the course of treatment at Aix-la-Chapelle. If this has already been tried, we know of no specific. In some cases counter-irritation along the spinal column has been tried, and said to be of use. Iodine liniment to scalp and spine has been recommended. Surgical treatment has been tried with the idea of relieving pressure, but the results of trephining have not been encouraging. I trust that sooner or later some antitoxin will be found which will aid in defeating this malign disease. Calabar bean was used for some time, but I cannot report favourably on its action; nux vomica is useful, with or without quinine and hydrobromic acid; I cannot say that setons in the neck have produced any good effect. In ordering drugs for general paralytics we have to remember that they are very readily affected by strong remedies; I have known minute doses of alkaloids produce in them very alarming symptoms. If there is much sleeplessness I prefer paraldehyde in drachm doses to any other drug; I do not like morphia or hyoscine; trional or sulphonal generally fails to produce sleep. If the patient is in fits we may use chloroform or nitrite of amyl; and nitro-glycerine, too, has been found of service. If the *status epilepticus* continue to an alarming extent, I advise an enema containing 20 grains of chloral with 20 grains of a bromide. It is necessary in these cases to consider details very carefully; thus it may be necessary to empty the bladder and perhaps to wash it out. Soreness of the skin may be prevented by great attention to keeping it dry, and also by the application, night and morning, of hot water and dry Castile soap.

f hæmatoma occurs it is best treated by immediate and vigorous listering; this failing, it may be necessary to slit the part open and emove the clot.

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INFANTILE CEREBRAL DEGENERATION

Syn.:—*Arrested Cerebral Development—Amaurotic Family Idiocy. A Family Form of Idiocy. Agenesis Corticalis.*

Definition.—A rare and fatal disease of infancy characterised by progressive mental and physical enfeeblement, associated with symmetrical changes at the macula lutea which are pathognomonic.

History.—Attention was first called to this disease by Mr. Waren Tay in 1881; and since then twenty-eight cases have been published, including Mr. Tay's patients. Ophthalmologists have contributed most of the cases on record, as the peculiar and characteristic ocular changes form a prominent part of the clinical picture of the disease. B. Sachs of New York, Peterson, Hirsch, and one of us (Kingdon) in this country have alone been successful in obtaining autopsies; the latter observer being particularly fortunate in that he has been able to supplement his clinical observations by autopsies and subsequent microscopical examination of the nervous system in three members of a family of seven, five of whom were affected.

Causes.—A curious circumstance, which appears to be of etiological significance, is that, of the published cases in which the nationality is stated, the patients have all been Hebrews. In addition to this racial peculiarity, there is distinct evidence that the condition belongs to the category of family diseases; more than one child in the same family is, as a rule, attacked, though others may remain perfectly healthy. Of the twenty-eight cases recorded, ten were in boys, and fourteen in girls; in four cases no mention is made of the sex.

Syphilis and other hereditary diatheses appear to play no part in its production; nor is there any evidence that consanguinity of marriage has any great influence in this connection, though it has been a possible factor in some cases. One or other parent has been neurotic in some instances, and injury during pregnancy has been noted, but this has been exceptional. No proximate cause after birth has yet been discovered, with the exception of a doubtful injury in a few cases.

Symptoms.—Children who afterwards show evidences of this disease are born at full term, are then, as far as can be judged, perfectly normal, and so remain until the end of the third (as a rule), or it may be as late as the sixth month; up to which time development progresses naturally. The first symptom to attract attention is an inability to sit up properly, and to keep the head from falling backwards; in addition to these evidences of weakness of the muscles of the neck and back, there is often a suspicion that vision is impaired; but usually no definite ophthalmoscopic changes can be made out so early.

As the disease progresses the child becomes apathetic, takes little notice of what is going on around it, and wears the vacant expression of mental enfeeblement. It becomes less and less able to sit up, and quite unable to hold up its head, which consequently falls backwards. It lies in bed as it is placed, and has no power of moving from one position to another. Objects placed in its hands are grasped feebly and soon dropped. The muscles become soft and flabby, but in the only instances in which they have been tested electrically in the early stage of the disease they responded to both the interrupted and constant currents. Soon there is evidence that the pyramidal system is implicated; slight extensor or flexor spasm appears in the limbs, and the tendon jerks become too active.

Later in the course of the illness the enfeebled muscles begin to waste; all the muscles of the body sharing in the atrophy, which eventually becomes extreme. To this muscular wasting, which is general and is confined to groups of muscles, there is added an increasing degree of spasmodic rigidity which causes retraction of the head, and distortion of the different segments of the limbs,—resulting, for instance, in inward rotation of the arm, pronation of the forearm, flexion of the thigh on the abdomen with marked abduction of the limbs, over-extension with partial luxation at the knee, extension of the foot, and so on. As the contracture increases the child evidently suffers much pain, which is increased by the spasmodic jerking of the limbs which occur from time to time. Death may result, however, before spasmodic contracture has taken place; or rigidity may have been occasional, no spasm being permanently present.

Convulsions are so rare that they form no part of the usual clinical picture of the affection, but have been observed as terminal manifestations of the illness. Cutaneous sensibility and the superficial reflexes are preserved, and the former has occasionally been found increased, so that the child starts at the slightest touch. The special senses, with the exception of vision and hearing, remain unimpaired throughout the illness. Sight fails gradually until total amaurosis results from optic atrophy, which has all the characters of being primary, and in the majority of cases is undoubtedly so; though Mr. Waren Tay has observed slight neuritis in the early stage of the optic nerve affection. In addition to this, ophthalmoscopic examination reveals a peculiar and distinctive appearance in the region of each yellow spot. At first a suspicious-looking haze is seen in the macular region, but subsequently a whitish gray oval patch is seen, almost twice the size of the optic disc, with softened edges, and with its long axis lying horizontally, very slightly raised above the general surface of the retina. The fovea centralis is seen as a dark cherry-red spot in the centre of this patch. Once established, these appearances remain unaltered throughout the whole course of the affection, and were present in Koller's patient, aged four years.

There is no evidence that either the cranial nerves—other than the second pair—or their nuclei are affected, speaking generally. The pupils are equal, and, as optic atrophy becomes advanced, they are dilated and inactive to light.

Strabismus has been noted exceptionally, as has nystagmus also; but both phenomena must be rare: towards the final stages of the illness, however, slow rhythmical movements of the eyes from side to side may occur. The only other points calling for comment, as far as the cranial nerves are concerned, are that hearing sometimes appears to be abnormally acute, a phenomenon too frequently observed to be merely accidental; and there may be difficulty in swallowing for some time prior to the fatal termination of the case.

Apart from any accidental complications, the thoracic and abdominal viscera are, as a rule, normal; the appetite good, the bowels regular, the urine natural, and the pulse, respirations and temperature normal. In Sachs' first case, however, the gastric mucosa early refused to perform its functions properly; and some elevation of temperature has been noted in the terminal stages of a few cases.

The disease has ended fatally in most of the recorded cases, and in those in which this result had not been reached, the condition of the patients at the time of publication made it evident that there was not likely to be any departure from the general rule; the only known exceptions being Koller's case of a girl aged four years, and a child aged four and a half years cited by Peterson. The duration of life varies from one and a half to two and a half years, is usually less than two years, and may (quite exceptionally) be prolonged beyond this, as in the two cases already referred to. The final stage of the affection sometimes resembles the same stage in cases of general paralysis of the insane. Death may occur before much emaciation, or evidence of spasmodic rigidity of the limbs. But usually the child becomes pale and shrunken, more and more lethargic and exhausted until marasmus becomes extreme, consciousness is lost, and death closes the scene. In exceptional cases, instead of this more usual form of death from marasmus and gradual exhaustion, the termination may be unexpectedly sudden.

Morbid anatomy.—The cerebral and spinal meninges usually present no abnormal appearances; though in Sachs' first case a few slight adhesions of the former were noted. The convolutions and sulci of the brain are normally arranged; but in the case just referred to there was some abnormal fissuration which was regarded as indicative of a brain of low development, an abnormality present in Peterson's case also. The sulci are wide in some cases, pointing to a certain amount of atrophy of the convolutions; but this atrophy varies in different cases. The ventricles are normal, and there is no excess of cerebro-spinal fluid in them; though there is some compensatory œdema of the meninges. The essential change met with on microscopical examination is degeneration of the neurons of the cerebral cortex, more especially those of the central convolutions. The pyramidal cells are seen in all stages of degeneration. Nissl's method of staining reveals various degrees of alteration in the size and shape of the cells; some are so distended as to become balloon-shaped, others preserve some traces of their original shape, while in some the changes are sufficiently advanced to lead to disintegration and vacuolation of the cell. The stichochrome granules disappear from the cells, and while, in some instances, fine chromatic granules are seen scattered through them, in others they are entirely without chromatophile substance. Phagocytes in the vicinity of the degenerating cells, and around the vessels, contain in their interior a large amount of the chromatophile substance which they have derived from the nerve-cells.

The Golgi-Cajal method of preparing portions of the cortex for examination also reveals clearly that degeneration of the neurons is in progress; in some cases the cells are variously altered beyond recognition, and all stages of degeneration are seen in the axons and dendrons. In most cases these processes are broken off from the cells, while, if still continuous with them, the characteristic beaded appearance of degeneration is seen along the course of the axis-cylinder. Evidences of similar changes are met with in other parts of the cortex, but they are not nearly so marked as in the region of the central convolutions.

By the Marchi method of preparation intense degeneration of the neurons may be traced through the corona radiata and internal capsule to the pyramids; this, and other methods of staining, reveal pronounced degeneration of the direct and crossed pyramidal tracts in the spinal cord on both sides. The perivascular lymphatics of the vessels in the corona radiata are full of fatty debris stained black by the osmic acid in Marchi's fluid. There is no evident change in the neuroglia; no sclerosis, no alteration in the walls of the blood-vessels, and no infiltration of the tissues in the neighbourhood of vessels. Degeneration of the cells of the basal ganglia, nuclei of cranial nerves, olivary bodies, and the anterior and posterior horns of the spinal cord have been found by Hirsch; the cells of the cerebellum alone showing but little change. Some degeneration has been found in the lemniscus on both sides, the descending roots of the fifth nerve and superior cerebellar peduncles. No changes have been found in the peripheral nerves.

The ocular changes consist in atrophy of the optic nerves, without any evidence of antecedent inflammatory mischief. Holden has found degeneration of the ganglion cells of the retina, otherwise nothing had been found to account for the changes seen in the macular region other than a thickening of the retina at this part, a change due to a spacing out of the outer molecular layer, as if by œdema (Treacher Collins).

Pathology.—While it is possible that the disease may be congenital in origin, neither in the clinical history nor in the morbid appearances met with after death have we any evidence of this. Nor do we find evidence that the affection is one of arrested development, as has been advocated by Sachs; for, while it is true that the changes met with lead to arrest of development and ultimate dissolution, it cannot be said that these changes in the central nervous system are themselves simply the result of arrest of development. On the contrary there is every reason to regard the changes as the result of a progressive degeneration of the neurons, such as might well result from the action of some toxin. The altered shapes of cells, regarded by Sachs as evidence of congenital maldevelopment, appear, as now investigated by more modern methods, rather to be the results of degeneration in normally developed structures; in none of the cases that we have examined has there been abnormal fissuration or other evidence of low cerebral development.

The advanced state of degeneration of the pyramidal tracts suggests the possibility that the changes in them precede those which are met with in the cerebral cortex; or that the changes in the cortex begin at a stage prior to the myelination of the pyramidal fibres. That the latter cannot be altogether the case is proved by the presence of many normally myelinated fibres in the pyramids, and of others in a state of recent degeneration with disintegration of their myelin sheaths. Again, that the former possibility is improbable is made evident by the fact that there is nothing in the clinical history of these cases to suggest that sclerosis of the pyramidal tracts precedes the affection of the cerebral cortex. There is no reason, therefore, why we should regard the morbid process otherwise than as one affecting the whole motor neuron from its beginning in the cerebral cortex to its end in the spinal cord; and, as the nutrition of the neuron depends upon the integrity of that part of it known as the cell, it is possible that the destructive process begins in the cell, and that the axon and dendrons suffer secondarily. On the other hand, the baneful influence may primarily attack the axon and dendrons; or all parts of the neuron may be affected simultaneously. Whether these changes depend on a deficiency or alteration in quality of some internal secretion, on the presence of some toxin, or on some other cause, remains for future research to determine. There is nothing in the histological changes to suggest an inflammatory process, syphilitic or other.

No satisfactory explanation of the relationship between the changes at the macula lutea and those met with in the central nervous system can be offered, unless it be that the ganglion cells of the retina are similarly affected to those of the cortex cerebri, and that they are thus affected has been shown by Holden. The occurrence of optic atrophy, on the other hand, is commonly met with in degenerative conditions of the central nervous system, though the mechanism of its production is not easy to explain.

Diagnosis.—When the changes in the region of the macula lutea are fully displayed they are so characteristic that any uncertainty as to the nature of the case can no longer exist. We know of no other condition in which progressive mental and physical enfeeblement are associated with these peculiar and pathognomonic changes. Even before the appearance of the macular changes the diagnosis is not difficult, though the affection may be then confounded with other infantile cerebral palsies. If a trustworthy history can be obtained, the pre-natal and natal affections are readily distinguished from this disease; the earliest evidences of which become manifest at the third month. It may be confounded with inherited syphilis; and the history of syphilis in a parent, and evidences of this disease, ocular or other, in the child must be sought for in attempting to arrive at a diagnosis. Ordinary congenital idiocy is distinguished by the absence of ocular symptoms and fundal changes, and such patients live for many years.

As regards its distinction from other post-natal cerebral palsies, and especially the family forms of these, reliance must be placed on the absence of convulsions; on the exceedingly gradual and general invasions of the paresis, which does not assume the form of a hemiplegia or a paraplegia; and, above all, on the presence of blindness, with the characteristic fundal changes. The possibility of confounding this disease with acute anterior poliomyelitis is too remote to deserve much attention; but should the question arise before the development of the changes in the eye grounds, the absence of altered electrical reaction of the muscles would exclude the spinal affection.

Prognosis.—With the exception of one case, recorded by Koller, in which the retinal changes were detected in an idiot aged four years, and the case referred to by Peterson, all the cases have ended fatally, and this usually within two years. Possibly as the condition becomes more widely recognised it may be found that more of the inmates of idiot asylums have been affected by this condition in infancy, and have escaped what appears to be the usual fate; but in the present state of our knowledge none but a gloomy prognosis is warranted. In the vast majority of cases as soon as the diagnosis is certain the prognosis is equally so. Moreover, the occurrence of a case of the kind in a family renders it highly probable that any children born subsequently may become similarly affected; fortunately this is not invariably the case.

Treatment.—No remedy appears to have any influence on the course of the disease, which is steadily progressive in spite of all the measures that have been tried for its relief. Mercury and iodide of potassium have both failed to influence its course; whether arsenic and similar remedies may prove more successful remains to be seen. If future researches should reveal any defect in connection with any of the ductless glands in these cases, the administration, in some form, of the particular gland affected might prove of service. So far the only measures of this kind that have been tried are the administration of pituitary gland and of cerebrine by one of us (Kingdon); both, however, with negative results. The same observer has made unsuccessful attempts to prevent the occurrence of the affection by administering iodide of potassium to the mother while pregnant. This was tried in two pregnancies; one child escaped, the other was attacked by the disease. We suggest that, in families where any case of this kind has been known to occur, the children should be weaned from the time of birth; on the chance that the mother herself, however healthy in appearance, may nevertheless communicate in her some deleterious product, chemical or other, which is capable of generating the disease in the suckling.

The most careful nursing, feeding and hygiene of such children are of course essential.

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THE CEREBRAL PALSIES OF CHILDREN

THE cerebral palsies of children are divided into two great classes—those in which there is evidence of the presence of paralysis when the child is born, and those in which, after a period, more or less prolonged, of apparently normal health and unimpaired activity, some form of paralysis appears. In the latter class the paralysis is, as a rule, hemiplegic in form; in the former both sides usually suffer: sometimes the legs only are affected, at any rate in a marked degree; sometimes both arms and both legs suffer; and in other cases one side only appears to be weak. Cases in which paralysis is present at birth are further subdivided into those in which the paralysis is the result of some difficulty or abnormality in the labour, and those in which it results from some condition arising during intra-uterine life; either primary disease or maldevelopment in the fœtus itself, or some morbid condition secondary to disease in the mother, or to injuries which she may have sustained during her pregnancy. By far the largest number are the result of abnormalities in the labour, the abnormality consisting usually in the length and difficulty of the labour, due to deformity in the mother or to malposition of the child. Nevertheless an unusually quick or precipitate labour may likewise give rise to birth palsy.

BIRTH PALSY.—**Symptoms.**—In this form of paralysis there is a variable distribution of weakness. Sometimes the lower limbs are affected alone, equally or unequally; producing the condition sometimes spoken of as "cerebral spastic paraplegia" or "congenital spastic paraplegia" ("Little's disease"). Sometimes the paralysis is of one side, affecting both arm and leg; sometimes both arms and both legs are affected; sometimes both legs and one arm only, or at least only one in a very obvious degree. In the limbs the weakness is usually associated with much stiffness. The gait, if the patient is able to walk, is characteristically spastic, each leg being dragged forward as a whole, and with considerable difficulty; if the upper limbs are affected there is, besides the weakness of the arms and hands, a great awkwardness in using them, probably occasioned by the spasticity. The knee-jerks are much exaggerated, but ankle clonus is not so frequently present as might be expected; and in the case of patients suffering from this condition it is usually present only in those who are able to walk. In patients unable to walk, in spite of the fact that a high degree of spasticity may be present, it is usually impossible to evoke it. In some patients the peculiar mobile spasm known as athetosis is present; and in patients in whom all four limbs are affected, athetosis affecting both sides—double athetosis—sometimes occurs. In this form spasm there is usually a cycle of movements, one position slowly and gradually passing into another. Sometimes the hand only is affected, and in the great majority of cases it is most affected; but the spasm may affect all the parts of the upper limb, and may also affect the leg. It involves the face in a few cases, and the platysma muscle may markedly participate in the affection.

Besides athetosis, or mobile spasm, another form of involuntary movement is sometimes present; a form very closely resembling the tremor present in cases of insular sclerosis, in that it is only induced during voluntary movement. This tremor is well marked when an attempt is made to carry out a definite movement with the affected limb. In carrying the affected hand to the face, for example, at the beginning of the act the tremor is very slight; but as the hand approaches the face it becomes more and more distinct and even violent, so that it is impossible for the patient to keep the hand in a position of rest. This condition has been unfortunately named "*chorea spastica*"; just as athetosis has been as unfortunately named "*post-hemiplegic chorea.*" In neither form of tremor is there, as a rule, anything more than a distant resemblance to the movements of true chorea; although certain cases of so-called hereditary chorea have undoubtedly been cases of birth palsy with double athetosis. But both athetosis and "chorea spastica" are probably more common in cases of infantile hemiplegia than in cases of birth palsy.

Paralysis or paresis of some ocular movements is also not infrequently met with; and this occasions a squint of varying character and intensity, apparently not the result of an affection of one muscle in one eye, but of a slight affection of the different movements in each eye. The sphincters of the rectum and bladder are only affected to the extent of being less easily controlled than normal. In patients in whom there is much psychical change, the calls are unattended to, and the habits are consequently dirty. Even in these cases, however, this is only the rule in the early years of life. There is rarely any affection of swallowing, although slobbering is not uncommon.

The *mental condition* varies much in different patients. In those in whom all four limbs are affected, there is, as a rule, much mental change: if the lower limbs only are paralysed the mental condition is frequently good; and in some instances the children are precociously sharp. When the paralysis is hemiplegic in form the psychical condition varies, being very defective in some, scarcely abnormal in others. In some cases in which the paralysis is only slight, much change is present; a result probably due, as will be pointed out later, not to the nature but to the position of the lesion. In all the cases mental development is retarded, and, as a rule, does not attain a high level.

Speech also is interfered with, and sometimes seems to remain entirely in abeyance; in nearly all, its development is retarded, and generally it remains long imperfect. Anything like true aphasia is, of course, scarcely to be expected, for this can only arise from interference with the functions of a speech mechanism already developed, and in cases of birth palsy we have to do with centres whose development has been interfered with, or entirely arrested.

Recurring convulsions are not so common in cases of birth palsy as might be expected, possibly because of the completeness and limitation of the destructive process which has been present. But in many cases a history of a severe convulsion is to be obtained, or more often of a series of severe fits, occurring a few days after birth. Occasionally such convulsions recur at intervals during the first few years of life, and in a small number of cases this tendency persists during the whole of life.

Deformities are apt to occur in consequence of the interference with development, and the malpositions encouraged by the form of paralysis. The most common deformity is some form of talipes; and, in patients in whom one side is weaker than the other, lateral curvature is almost inevitable, unless extreme care be taken.

Causes.—In the great majority of the cases of birth palsy, as I have said, the lesion is determined by some fault in parturition. As a rule there is a history of a long and difficult labour, in many cases the result of an abnormal presentation of the fœtus; and frequently of a labour necessitating the use of instruments, or of turning. Often also the child is born cyanosed, and not breathing; so that artificial methods of promoting respiration have to be resorted to. Not uncommonly also the child is born before full term. But, as already pointed out in reference to these cases, any injury to the child occurring in a labour in which instruments have been used, or in which operative interference has been resorted to, is not to be ascribed to the instruments used or the operation performed, but rather to those abnormal conditions which rendered interference necessary. Besides the long and difficult labour, the quick, or, in technical language, the precipitate labour also seems to have an effect in the causation of the condition under consideration.

As abnormal labours are much more common in primiparæ than in multiparæ, it is accordingly to be expected that birth palsy will occur much more frequently in first-born children: this is actually the case in so large a majority of instances as to place the fact outside the region of mere coincidence. Two other facts come out in a series of cases of this character; namely, that of children affected with birth palsy there are twice as many males as females; and that the child of an elderly primipara is more liable to be afflicted than is the first child of a younger woman. These two facts lend support to the view which ascribes the great majority of cases of birth palsy to mechanical difficulties in the process of parturition; for in the former case the larger size of the child, in the latter the greater rigidity in the maternal parts will tend to increase the difficulties of the labour.

There is some evidence that inherited syphilis is sometimes a factor in the production of this condition. It is very uncommon to see a child the subject of birth palsy who has any of the recognised signs of congenital syphilis; but it is quite possible, of course, that a syphilitic child may suffer from birth palsy if it has been exposed also to the causes of this condition; and it is likewise probable that inherited syphilis, through affection of blood-vessels or membranes, may occasionally give rise to a morbid process which causes in the child symptoms similar to those which we have under consideration.

Morbid anatomy.—There is little doubt, from the cases which have been examined after death, that the condition upon which the paralysis depends is, in most cases, hæmorrhage occurring under the dura mater, and so affecting the cortical structures as to abolish or interfere with their function. From what has been said as to the causation, it will be seen that such a condition is what we should expect; and there seems no reason to doubt that its occurrence is due to the rupture of vessels in consequence of the mechanical difficulties in the act of parturition. The situation of this hæmorrhage gives the clue to the distribution of the paralysis. As a rule, as I have already said, the legs are more affected than the arms, sometimes they are affected alone. In such a case the hæmorrhage will be found on each side of the longitudinal fissure, spreading in each cortex over the area in which the leg movements are chiefly represented. If we suppose the hæmorrhage on one side to spread further, so as to destroy or impair the area in which the arm movements are represented, a condition would be produced in which both legs and one arm would be affected; if the hæmorrhage spread still further, one side of the face would be affected; and if it spread on each side, so as to affect both arm areas as well as the leg areas, a condition would be produced giving rise to paralysis of both arms and both legs. In cases in which the speech function is in abeyance, either completely or partially, the lesion is of the third frontal convolution on both sides or only on one; and it will readily be understood that the ocular movements may be interfered with if the lesion spread forward so as to involve the area related to these movements; thus a variable squint may be produced. I repeat that a varying degree of psychical defect is found to occur in children the subjects of birth palsy; and it may reasonably be assumed that this depends upon injury to that part of the brain which is probably most intimately associated with psychical processes—the præfrontal area.

This morbid condition is only distinctly recognised in cases in which death occurs in the early stage. Such, however, are not common; and when death takes place after the lapse of some years, the condition discovered varies considerably. Sometimes it is what is known as porencephalus, a condition in which there is much cystic formation surrounded by cerebral substance more or less altered: sometimes there seems to be a sclerotic condition of the brain itself, and in other cases a condition resembling pachymeningitis, due to the effused blood having become organised and altered. In short, from the condition found in such cases it would be impossible to discover the nature of the original lesion, were it not for the information gained from the examination of recent cases. It is also held by some writers that in certain cases, those especially in which the child is a premature one, the resulting condition is due to incomplete development of the pyramidal tracts. In such cases it is said that gradual improvement usually follows, and may go on to allow of an almost normal power of locomotion.

Diagnosis.—The diagnosis of the condition will not, as a rule, present many difficulties. The onset at birth, although it may not be noticed until a few days after it, and usually the history of some difficulty of abnormality at birth, or at any rate that the child was the first-born, are the points on which a diagnosis will depend. Infantile hemiplegia may be similar in the physical condition, or in the psychical symptoms; but in this disease the onset is later, it comes on in a previously healthy child, it is ushered in with a convulsion or convulsions, and it is strictly hemiplegic. The spastic paralysis due to caries may closely resemble cerebral spastic paralysis; but in the former condition there has been a period during which the limbs were used normally. Of infantile paralysis also—acute anterior poliomyelitis—the same may be said; and in this condition the muscular wasting, flaccidity, and loss of reflex action and of faradic irritability in the muscles will sufficiently distinguish it.

Prognosis.—Such a condition as that described entirely precludes any hope of complete recovery. The degree of recovery which takes place naturally depends on the extent and situation of the lesion; and will be greater when this is limited to the leg areas on each side of the longitudinal fissure. A patient with this condition will be late in acquiring the ability to walk, and even at the best will always walk stiffly and imperfectly; but the arms will be useful limbs, and the mental and intellectual powers will not necessarily be impaired at all, may even be rather above the average. In cases in which the upper limbs are affected there is, as will be evident from what has already been stated, a much greater likelihood of mental impairment accompanying the bodily ailment; and where such mental defect is present the improvement will not be great, at any rate in reference to that particular symptom.

But physical improvement takes place in all these cases. Often, although the children attain the age of five or six before they can walk, many of them, in whom the prospect of walking seemed out of the question in an early stage, do ultimately walk, imperfectly it is true. In short, physical improvement takes place in all, and the degree of this can only be estimated by ascertaining actually how much impairment is present; mental improvement also takes place even in children apparently quite imbecile at first. But in cases in which mental impairment is present, a normal mental condition must not be hoped for, any more than a perfect physical condition is to be expected in any case.

Treatment.—A child affected with birth palsy naturally requires very great care. Good and intelligent nursing is essential; suitable food and warm clothing are even of more importance than in the case of ordinary babies. No effort should be spared in attempting to develop the physical and mental capacity; for much may be done in this way to increase its powers. But such training is apt to be discouraging because of the slow, almost invisible progress which takes place, and it is well, therefore, that the nurse should not expect very rapid progress. Gymnastic exercises, such as will assist in increasing muscular power, rubbing of the limbs and passive exercises are of much importance. Cod-liver oil, cream, and fat in an easily digested form are to be regarded as articles of diet rather than as medicines.

Attention also will be given to measures calculated to prevent deformities, such as talipes or lateral curvature; or, if they have already occurred, to the operative or mechanical treatment which will tend to correct them, or at least to minimise their effect.

INFANTILE HEMIPLEGIA.—Infantile hemiplegia is the name applied to that paralysis of one side of the body which occurs in the first six years of life. Although in its general symptoms, except those occurring at the onset of the attack, it closely resembles the hemiplegia of adults, yet there are some distinct differences. The majority of these, however, are to be referred to the early age at which it occurs, and the consequent interference with development both of the brain and of the affected limbs. A distinction must be drawn not so much as regards symptomatology, but in reference to pathology, between cases of infantile hemiplegia proper, which is probably a peculiar disease, and cases of hemiplegia occurring in children in connection with acute disease; especially with rheumatism, scarlet fever, diphtheria, or typhoid fever.

The hemiplegia of infancy occurs during the first six years of life, but much more frequently in the first two years than later. Of forty-two cases, nineteen occurred in the first year, sixteen in the second, three in the third, three in the fourth, and only one in the fifth. The illness begins as a rule with general malaise and feverishness, and this culminates in a severe fit, or a series of convulsions. These are generally unilateral, but may spread so as to affect both sides. They may persist with but slight intermission for several days; and after the fits have ceased it is found that the child is paralysed on the side on which the convulsion was most severe, or to which it was limited. The resulting paralysis is permanent, although in degree it may be modified; and as a rule a considerable improvement from the condition immediately subsequent to the fit is to be looked for. If speech were present at the time of onset of the fits, it is usually interfered with at first, being either completely lost or rendered much less perfect than it had been before. Anything like the true aphasia of adult life is rarely if ever present; and it is doubtful whether the name aphasia is strictly applicable to the condition which results. Permanent speechlessness occurs in some cases, even in children who had already acquired the faculty of speech more or less imperfectly. Permanent aphasia practically never occurs.

As a rule then there is, after the convulsion or series of convulsions is over, paralysis of one side of the body affecting face, arm and leg. The facial affection is usually slight; rarely permanent. The arm is generally more affected than the leg; the knee-jerk is exaggerated, and ankle clonus is of frequent occurrence. Rigidity is not usually so extreme as in adult cases; but some features exist in infantile hemiplegia which are at any rate much less common in adults. Amongst these probably the most striking is what is sometimes spoken of as atrophy of the affected side. But this is obviously an erroneous term to use, the condition is more correctly described as maldevelopment of the affected side. In a large proportion of cases the smaller size of the structures on the affected side is striking. This defect in development affects not only muscles and other soft structures, but, in a well-marked case, the bones also are much smaller than the corresponding structures on the unaffected side. The arm is usually more strikingly affected than the leg; and this is in accordance with the usual distribution of the paralysis. Not infrequently when the defect in development is so slight as to be doubtful, a comparison of the two shoulder-blades will make it evident that one is distinctly larger than the other. In rare cases only is the difference in size between one side of the face and the other distinct and striking. The dependence of this feature of infantile hemiplegia on the central lesion is probably indirect. The defective development of the structures on the affected side is more probably the result of the disuse which the paralysis enforces, than of any direct neurotrophic influence.

Another condition found in infantile hemiplegia, not so frequently as to be pathognomonic but much more frequently than in other varieties of hemiplegia, is a form of tremor, the so-called mobile spasm, or *athetosis*. This only occurs in a small proportion of cases of infantile hemiplegia (probably about 10 per cent); but it is much more frequent than in hemiplegia occurring in the adult. Indeed, its occurrence in adults, unless the hemiplegia date from an early age, is very uncommon; and in most of the cases in which its onset has taken place during adult life it has been in patients in whom the paralysis was the result of accident. Its presence may lead to hypertrophy of certain arm muscles. This form of movement has been already described under birth palsy. In that section also a curious form of tremor, sometimes named "chorea spastica," closely simulating the tremor of disseminated sclerosis in that it is evoked only during voluntary movement, has been mentioned. Of forty-two cases of infantile hemiplegia two manifested this peculiar tremor; but probably it does not occur nearly so frequently as this proportion would seem to indicate.

As I have said, this condition is usually ushered in with a series of fits beginning on the side subsequently paralysed. Frequently these fits recur at intervals, sometimes short, sometimes prolonged. They are, as a rule, unilateral, affecting the paralysed side. Some observers, however, have stated that the fits affect the non-paralysed side. It is conceivable that this may be the case if the paralysis is severe; while the paralysed side may be that affected in the fits only when the paralysis is less complete. The fits are usually accompanied with loss of consciousness; and consist of tonic and clonic spasm, with occasional tongue-biting, and relaxation of the sphincters. But sometimes the attacks are of the nature of petit mal, and consist in momentary loss of consciousness without convulsion. The cases in which this occurs are cases in which the paralysis is slight. In one the attacks, which occurred frequently, were succeeded by the condition known as automatism, in which the patient, while still unconscious, performed apparently purposive acts. Such a condition is, of course, not only of great interest, but of the highest medico-legal importance. [See later art. on "Epilepsy and Insanity."]

Besides the evidences of interference with physical functions, in a certain number of cases there is some mental deficiency. Often this is considerable in cases in which the physical functions are but slightly interfered with; and in such cases the mental disturbance not infrequently assumes the form of restless and irresponsible activity. In other patients a dull and apathetic condition of dementia is present; and in them the physical condition is frequently one of considerable, sometimes profound, unilateral weakness. But no constant relation is to be found between the physical and mental impairment, and where the latter exists there is probably mischief in the præfrontal region. It is easy to imagine a condition in which it would be considerably affected without any very noticeable degree of interference with motor functions.

Besides these cases of what may be called infantile hemiplegia proper, other cases of hemiplegia of cerebral origin are met with in children, occurring after acute diseases; especially scarlet fever and diphtheria. Such a condition is the result of some intercurrent complication of these diseases. In the former disease it is probably due to embolism, a result of associated endocarditis; in the latter the same process has been alleged, but in four cases seen by myself there was no evidence of any endocarditis; and thrombosis or hæmorrhage is to be regarded as a more likely cause. Hemiplegia also sometimes follows typhoid fever in a child. As blocking of veins in other parts is not uncommon in this disease, a similar process in the cerebral veins is to be regarded as the probable cause of the palsy. The hemiplegia in the cases of this nature is not generally associated with fits, nor with any profound psychical change; but is accompanied by maldevelopment of the limbs on the affected side. In regard to diagnosis, prognosis, and treatment, what is to be said in reference to ordinary cases equally applies to cases of this nature.

Pathology and Morbid anatomy.—The ultimate pathology of this condition is still largely a matter of conjecture. There does not seem to be any constant relation of season to the onset, as is the case in Infantile paralysis; but it must be remembered that these cases are usually seen when the paralysis has already existed for years, and when the recollection of the conditions attending its onset has become indistinct and untrustworthy. Even the morbid anatomy is still uncertain, for few patients die, or at any rate are examined after death while the condition is recent; and the state of the brain after a time is such as to give little or no clue to its initial morbid state. Two views are held with regard to its morbid anatomy; according to Strumpell the condition of infantile hemiplegia is a result of an acute inflammation of the gray matter of the cortex, analogous in some ways to that in the gray matter of the spinal cord which underlies acute anterior poliomyelitis; this condition he names *Acute polioencephalitis*, and if his view be accepted the ultimate pathology would probably have to be looked for in some infective process. The other view regards the condition of the nervous system as secondary to some vascular obstruction; and this, according to Sir W. Gowers, is to be looked for in the veins of the surface. No definite data are yet available to decide between those rival hypotheses. The condition which has been found in the cases hitherto examined has been one either of cystic formation (*porencephalus*) with much atrophy of the affected hemisphere and of the correlated parts of the nervous system, or one of sclerosis of the hemisphere; and both these conditions are, of course, compatible with the initial lesion formulated in each hypothesis. Nor can anything definite be said as to the cause of the athetosis present in a number of the cases. It is probably a result of imperfect destruction; but whether in the cortex or in the deeper structures it is still impossible to say. Even the case published by Beevor and Horsley, in which removal of a cortical area in an adult brought about a cessation of the athetosis, is not conclusive.

The **diagnosis** is not ordinarily difficult. A case of birth palsy may closely simulate the clinical condition of infantile hemiplegia; but the history of weakness actually at the time of birth, although only first noticed a few days later, will usually be sufficient to indicate the category to which a given case is to be referred. From infantile paralysis the state of the reflexes and the absence of any electrical change in the muscles of the affected limbs will sufficiently serve as a distinction. Some cases of myopathy may present a superficial resemblance; but the hemiplegic character of the affection, and the presence even of excess of the knee-jerk, as well as the history, especially of convulsions, will usually be sufficient indication to allow of a correct diagnosis being made.

Apart from the danger which the weakness and the liability to convulsions imply, the **prognosis** is good, so far as life is concerned. It is also found that the hemiplegic weakness is usually greater immediately after the onset of paralysis than it is ultimately; so that we may predict a certain degree of improvement. But the weakness will remain: the child will probably be backward, and may have a definite degree of mental weakness. The fits can usually be controlled if the patient is carefully, judiciously, and persistently treated. This, however, is only partly true of cases in which petit mal is present. But even in cases in which no fits have occurred, except the one convulsion or the series of fits at the onset, it must always be remembered that fits are prone to occur, although their occurrence may be delayed even for as long as several years. If speech has been impaired the impairment is rarely if ever persistent, and sensory impairment is not found, although hemianopsia has been described.

Treatment.—Great care and patience must be exercised in cases of mental backwardness to develop the existing mental power cautiously and judiciously. The same is true of the defective physical power; and mild gymnastic exercises and passive movements will do much to increase the power which the patient retains. Massage will also assist in minimising the rigidity; but electricity is not of much use, and its application may do harm by causing an amount of discomfort and alarm quite out of proportion to any benefit likely to be derived. Convulsions will be treated like epileptic fits. They are usually amenable to ordinary treatment by bromides; but the attacks are all or mostly attacks of petit mal, which are much more intractable. Fresh air, good food, and the best possible hygienic conditions are, of course, essential.

MICROCEPHALY.—This condition deserves a brief notice. Cases of different kinds have been gathered under this name, but, so far as can be ascertained, only two varieties need be considered; namely, those in which the small or deformed skull is secondary to actual brain disease, such as the birth palsy, or infantile hemiplegia just considered, and those in which the smallness of the skull and of the body generally is but a local expression of a general developmental defect in which the brain itself shares. The cases of the former class may be characterised by some local weakness or paralysis; in those of the latter the weakness is universal: in both classes the mental state is very backward. In the last few years operative treatment has been recommended in such cases, the object being by removal of the cranial wall to relieve a hypothetical condition of pressure and consequent interference with brain growth. No results, however, have been obtained to justify such procedures, and the cases cannot be regarded as in any degree amenable to treatment other than the educative and disciplinary influences resorted to in the treatment of weak-minded children.

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J. T.

OTHER DISEASES OF THE NERVOUS SYSTEM

DISORDERS OF SLEEP

INSOMNIA—SOMNOLENCE—DREAMS AND NIGHTMARE—NIGHT-TERRORS OF CHILDREN—SLEEP-TALKING—SLEEP-WALKING.

Physiology.—It is unnecessary in this article to enter into a discussion on the physiology of sleep. Certain symptoms—diminished metabolism, shallower respiration, less frequent pulse, diminished secretory activity, and loss of consciousness—are recognised as constant accompaniments, but a satisfactory explanation of the condition is not yet found. There are several hypotheses as to the causation of sleep. It has been attributed to cerebral anæmia; to chemical changes in the brain cells or neurons, such as an exhaustion of their intramolecular oxygen, or an accumulation of fatigue products; to a contraction of the dendritic processes, and a consequent break in the transmission of nervous impulses; to an expansion of the neuroglial cell processes insulating the nerve-cell processes, and producing the same effect; and to a purely psychological condition, namely, loss of consciousness apart from any physical or chemical change. The last explanation is simply a cloak for our ignorance. The most probable hypothesis is that of an altered metabolism of the cerebral cells dependent upon exhaustion and diminished influx of stimuli.

Anæmia, or relative anæmia, is an important factor, according to many authors the most important. As the vascular contents of the cranial cavity must remain practically constant, any loss of blood in the arterioles and capillaries must be compensated by some increase elsewhere. According to Cappie, this occurs in the pial veins; according to Howell, in the veins at the base of the brain. In any case there is a relative anæmia—a diminished blood-flow through the cerebral cortex, and consequently a diminished metabolism and depressed function; but the bearing of this upon the production of sleep we do not know. Howell believes it is important. From plethysmographic observations on the cutaneous circulation, he was led to suppose that exhaustion of the vasomotor centre is the all-important condition; but this opinion has been strongly criticised by Dr. Leonard Hill. As far as we can see, neither the circulatory, nor the histological, nor the psychological hypotheses are sufficient to account for sleep; and, with the exception of the circulatory, none of them bears any practical significance. The chemical hypothesis, on the other hand, is of direct importance, as it explains in some measure why, in order to induce sleep, it is necessary to rid the organism of fatigue products and toxins.

Amount of sleep.—The amount of sleep required by a healthy person depends upon his age, the quality of the sleep, and his idiosyncrasy. A baby sleeps the greater part of his time; as the child grows up less sleep is needed; and at the age of fifteen, nine to ten hours are usually thought enough (*vide* vol. i. p. 470). Between fifteen and twenty-five years of age eight hours should be allowed; afterwards the amount must depend upon the work and requirements of the individual. Some people can do with from four to five hours; and others with even less than this amount, as was the case with Brunel and Alexander von Humboldt; for the majority of people, however, from six to seven hours are usually enough. Idiots and persons of weak intellect often sleep excessively, and even in more normal individuals a condition of abnormally prolonged sleep is occasionally recognised. Old people also require an increased amount of sleep. When sleep is profound and continuous, less is required than when it is broken and superficial.

INSOMNIA.—By this term is meant loss of the normal amount of sleep. It includes all grades, from simple restlessness to total loss of sleep. From what has been said it is obvious that the term is comparative; that what is sleeplessness in one person is not so in another. Furthermore, the kind of sleeplessness varies. Some people go to sleep directly after getting into bed, but awake every hour for several hours, sleeping well the rest of the night; others, especially the gouty, awake punctually at three or four, and are unable to sleep again, or, at any rate, until it is time to get up; others again find great difficulty in getting off, but, once asleep, do not awake until the morning. Some persons, as we have said, do not sleep at all, and these cases are of serious omen. It has been stated that sleep is more necessary than food; that animals die sooner from sleeplessness than from starvation; yet patients occasionally assert that they have not slept for weeks. In most cases this is probably illusive: they sleep without realising it.

In general, sleeplessness cannot be considered a disease in itself; it is, however, an important symptom of many diseases, and in certain cases, especially of neurotic women and over-tired men, it is the predominant one, and calls for careful treatment, otherwise it may lead to grave consequences. As the essential feature in the treatment of this condition is to discover, and, if possible, to remove the cause, it will be convenient to consider insomnia with regard to (*a*) its causes, (*b*) its treatment.

Causes.—These are many, hence it is necessary to adopt some principle of classification. This has been done by most authors, but no system has yet received general acceptance. Germain Sée recognises nine divisions: dolorous, digestive, cardiac and dyspnœal, cerebro-spinal and neurotic, psychic, insomnia of cerebral and physical fatigue, genito-urinary, febrile, and toxic. Others admit only four or five. We shall adopt a division into four. But it must not be assumed that all causes can be uniformly arranged in these classes; in some cases the immediate cause will fall under two or more divisions. Such divisions are of course only justified for descriptive purposes.

1. *Irritative causes.*—This class includes all forms of insomnia caused by pain and like uneasiness. In children, teething, indigestion, worms, and so forth, are frequent causes. Eye strain—as in hypermetropia and astigmatism, the irritation of an eczema, the inconveniences produced by faucial adenoids, and other similar conditions, are also etiological factors of insomnia. After surgical operations, even in the absence of pain, insomnia is not infrequent; this may be due in part to the constrained position of the patient and some irritability of the wound, in part to toxæmic and psychical causes. Cold feet, vesical affections, asthma, bronchitis, and other diseases accompanied by troublesome if slight irritations, for example, cough, or pruritus (local and general), also fall under this head. But the most important causal factor of this group is undoubtedly pain. Of all diseases accompanied by pain, as neuralgia, sciatica, migraine, angina pectoris, aneurysm, locomotor ataxy, the various forms of colic, acute inflammation of the serous membranes (peritonitis, pleuritis, pericarditis, meningitis), cerebral tumours and abscesses, malignant growths in various parts, diseased bone, acute and chronic rheumatism, osteo-arthritis, syphilis (the pains of which are often worse at night), and acute gout, sleeplessness is a symptom. The insomnia of all or many of these may involve other etiological factors, but the pain is the predominant one, and that which necessitates treatment.

2. *Toxic causes.*—A large number of diseases are due to the presence of some toxic agent in the blood, or are accompanied by it; and such diseases are often attended by troublesome insomnia. Alcoholism and nicotism, the exanthems (measles, small-pox, scarlatina), enteric and other continued fevers, erysipelas, diphtheria, pneumonia, influenza, hepatic disorders (the lithæmia of Murchison), dyspepsia and other gastric and intestinal disorders, rheumatism, gout, and Bright's disease are some of the conditions which fall under this head.

The insomnia accompanying heart disease and altered vascular conditions (arterio-sclerosis, etc.) may also be included in this division, as their origin in most instances appears to be a blood poison. This form of insomnia also is due in part to a deficient blood-supply, and possibly to an altered cardiac action. The sleeplessness induced by certain beverages, such as tea, coffee, and cocoa, requires mention here. These, especially if taken strong and late at night, are in most people powerful preventives of sleep. Strychnine and other nervine tonics, when taken regularly, often cause sleeplessness during the earlier part of the night; but in certain conditions of exhaustion, by improving the circulation and other functions, they prove excellent hypnotics.

Insomnia is also a symptom of certain drug habits, such as opium-eating, morphinism, and cocainism.

3. *Psychical causes.*—Grief, shock, worry, and mental anxiety are among the most frequent causes of insomnia. In many persons a predisposing factor—a nervous temperament, neurasthenia, hysteria, hypochondriasis—also exists, and among such persons insomnia may easily be established as a habit. In students, overwork, especially if accompanied by irregularity in retiring to rest and the prospect of an examination, are the most frequent causes. Women at the menopause often suffer from insomnia, partly, perhaps, owing to the accumulation of toxic products not eliminated by the catamenia. The various forms of insanity—such as mania, melancholia, general paralysis—are accompanied by insomnia: in some cases it appears as a premonitory symptom; although there can be no doubt, on the other hand, that continued loss of sleep is also a factor in the production of mental aberrations. Cases of severe chorea and of paralysis agitans are also marked by sleeplessness.

4. *Causes arising from change in the mode of life.*—Eating late dinners by those unaccustomed to them, and change of climate, especially to high altitudes, or in some individuals even to the seaside, sometimes give rise to temporary insomnia. Nurses who have been on night duty, after changing to the day frequently suffer from sleeplessness; and the same condition occurs in other people with intermittent nocturnal occupations. Sometimes very simple changes in the mode of life of individuals will lead to insomnia. A patient of mine and his wife both slept very badly for the first three months after beginning to cycle. This may have been due to excessive fatigue, or possibly to increased waste products in the blood, or to both combined.

Pathology.—Upon this subject we know very little; and so long as the physiology of sleep is ill understood, the pathology must remain unknown. As we have seen, the condition of the circulation and the composition of the blood are important factors in the production of sleep, and when the brain cells are improperly supplied with blood (as in anæmia and heart disease), or with impure blood (as in the various toxæmic diseases), we can readily understand that the nutrition of the nerve-cells is altered, and that some deviation from normal sleep must occur.

Treatment.—This naturally falls under two heads—general or non-medicinal, and medicinal—although these cannot well be separated in practice. In all cases the first thing to look for is the cause, so that, if possible, this may be removed. If it be an indigestion, this should be treated. Acid dyspepsia and that attending gout are frequently accompanied by insomnia. In these cases, careful attention to the diet, the avoidance of tea and fruit (cooked and uncooked), and of all substances containing vegetable acids, except in so far as these may be used in treatment, are essential. Fruit, owing to the acid it contains, is, I believe, an unsuspected cause of indigestion, gout, and sleeplessness in many persons: so also are acid wines. For gouty persons much butcher's meat, especially beef, should be prohibited, and white fish, poultry, and game ordered in its place. Fried fat, such as that on the outside of fried fish, should also be avoided. An alkaline bitter before meals, an occasional mercurial at bedtime followed by a saline aperient the next morning, and, for the gouty, colchicum or sodium salicylate should be prescribed. Sometimes a dose of Gregory's powder at bedtime, or a mild carminative, is of value. An overloaded colon is a common cause of sleeplessness in elderly people; for this an evacuation of the bowels at bedtime is the best means of relief. One of my patients found that a little peppermint water taken at bedtime gave him a better night than any hypnotic. In the insomnia of the aged and the overtired, and in that due to cardiac and vascular disease, strychnine and nux vomica are beneficial remedies: they tone up the circulatory system, remove indigestion and flatulence, and act directly on the cerebral cells. In cardiac cases and in neurasthenics with low arterial pressure, digitalis and the other cardiac tonics may be resorted to. In anæmia, iron, nux vomica, and sometimes digitalis are required; and similarly other diseases which are causal factors of insomnia must be treated in a more or less specific way. The appropriate remedies for each disease are described in other parts of this work.

In all cases of insomnia certain general precautions must be taken. The bedroom ought to be in a quiet part of the house, well ventilated, and of moderate temperature. Light should be carefully excluded, and the apartment scantily furnished. The bed should vary according to the habits of the individual; for young and middle-aged adults a firm mattress is the best, but for the old a softer bedding may be necessary. The covering should be light and warm; but in the use of pillows no general recommendation can be made. Some people sleep better with the head raised, others prefer to keep it on a level with the body. In heart disease it will be found necessary to raise it; but as a rule this precaution may be left to the choice of the individual. For broad-shouldered people Whitla recommends the wedge-shaped pillow used by the Germans. Invalids find much refreshment in the ultimate use of two beds for day and night.

For ordinary cases of sleeplessness simple means very often suffice. Some people read themselves to sleep, some count, others, like Southey, think of some monotonous discourse. One of my patients used to hang his feet out of bed for some time and then put them in again. Walking about naked, or a cold or tepid bath is often useful. Massage, especially of the abdomen, thighs, and legs, as in Dr. Eccles' method of treating insomnia, is sometimes advantageous. This method is believed to produce temporary anæmia of the brain, by causing a determination of blood to the manipulated parts; and it may be further aided by a hot compress to the abdomen. In the case of cold feet, vigorous rubbing, or a hot bottle, or a hot footbath with mustard in it, is beneficial; or again a hot sitz-bath may be used. Attention should also be given to the work of the stomach. As a rule a light supper is the best, and for many, and especially those who awake in the middle of the night, a little hot milk or meat juice containing a small amount of alcohol is helpful. The evacuations should also be attended to, and the bladder especially should be relieved.

Sleeplessness from overwork, especially from literary work, requires mental rest and change of air and scene. Temporary exposure to the cool air of the bedroom, or the wet pack, or a bath is often of use; but if the insomnia continue, it is necessary to give a mild hypnotic, such as twenty grains of sulphonal or trional, or thirty or forty grains of bromide of potassium, to break the habit of sleeplessness. Capsules containing ♏xxx. of turpentine given at bedtime are sometimes beneficial in the insomnia of overwork and worry. The drug acts as a stimulant and derivative, and is stated to succeed best in plethoric cases. No beverages containing caffeine should be taken after breakfast.

In nervous and hysterical women, and especially in women at the menopause, the bromides are very useful. I have long been in the habit of giving a mixture of bromide—either of potassium, sodium, or ammonium—tincture of sumbul, and tincture of hop, in camphor water, at the climacteric; and it has helped to remove the insomnia as well as the mental depression and flushing heats so common at this period.

The sleeplessness of the insane requires careful management. In the early stages of acute mania the bromides, chloral, hyoscine hydrobromide, and other sedatives are useful; but a hot bath at a temperature of 104° F., and cold water simultaneously poured upon the head are most efficacious in inducing sleep. In melancholia, where arterial pressure is usually high, paraldehyde in doses of ♏xl. to ♏xc., or even more, is a valuable hypnotic; so is morphine; but a 1-grain dose of erythrol tetranitrate, by reducing blood-pressure, will frequently act better than anything else.

In mild cases of delirium tremens sleep usually comes on after a time, whatever treatment be adopted; in the more severe cases chloral and bromides, alone or in combination, are beneficial. Paraldehyde is recommended by some physicians. Opiates may be given, but in most cases hyoscine is probably a more efficient remedy. Among the medical officers of the United States army 20 grains of powdered capsicum in the form of a bolus is the favourite hypnotic in this complaint. I have no practical experience of this prescription, and cannot therefore express any opinion of its value as a mode of treatment. Cerebral depressants should be given as little as possible, and the treatment should be confined chiefly to feeding and tonic measures.

In pneumonia sleep comes usually at the crisis; but where this has not occurred I have occasionally seen a hypnotic, such as chloralamide or paraldehyde, turn the scales in favour of the patient.

In pleurisy, and most other serous inflammations, 5 to 10 grains of Dover's powder usually conduce to sleep; mainly by relieving the pain. A hypodermic injection of morphine may be given with the same object in view.

In bronchitis, chloral and chloralamide are safe hypnotics; as a rule opiates are to be avoided.

The sleeplessness of asthma is relieved by remedies which cut short an attack, such as chloral hydrate, the fumes of Himrod's and other anti-asthmatic powders, the hypodermic injection of morphine, or, in some cases, a dose of 5 to 10 grains of citrate of caffeine. Bromides are also useful and so is paraldehyde, which both relieves the asthma and causes sleep. A change of locality, if only to another part of the same town, often succeeds. In one case, the removal of a student from Downing College, Cambridge, to a house across the street brought relief; and in another of my pupils the change from Caius College to a house in another part of the town brought to an end a most troublesome attack of asthma.

The insomnia of heart disease is benefited by digitalis, strophanthus, strychnine, and other cardiac tonics; but in some cases it is necessary to resort to morphine, either by the mouth, or still better, hypodermically, as first suggested by Professor Allbutt. Paraldehyde and chloralamide are most useful in my experience; they are less depressing to the circulation than chloral hydrate. Ice to the head is recommended by Dr. A. Morison, where the vital forces are not too low, or the temperature subnormal. It often produces sleep rapidly, with a more regular cardiac action. Heat may possibly answer in other cases presenting a subnormal temperature.

In chronic Bright's disease insomnia is occasionally very troublesome. Eliminants, such as aperients, should be tried, and if they do not succeed chloral hydrate may be given; it is a safer drug in kidney than in heart disease, the accompanying reduction of blood-pressure being usually beneficial. Morphine and hyoscine hydrobromide subcutaneously injected have been recommended in obstinate cases; but their employment requires great caution. Erythrol tetranitrate, by reducing blood-pressure, often acts like a charm even when sedatives have failed; and in one of my patients thorough rubbing of the skin by means of a flesh-brush induced sleep, and very materially relieved the restlessness of this complaint. In the sleeplessness dependent upon cirrhosis of the kidney Dr. Nestor Tirard recommends hyoscine, sulphonal, and paraldehyde; he is opposed to the use of opium and morphine.

When pain is the causal factor of insomnia morphine is the best general remedy, and it should be pushed until relief is obtained. In cases of neuralgia, locomotor ataxy, and so forth, some of the synthetic analgesics—phenazone or phenacetin—are of value. These drugs act also, I believe, as hypnotics in cases where there is no pain.

Calcium chloride is a valuable remedy in the insomnia due to pruritus.

This article would be incomplete without a more detailed reference to some of the more important hypnotics. These are comparatively few, although the number of the drugs of this class which have been advocated from time to time is large.

The *bromides* of potassium, sodium, and ammonium are mild, safe, and trustworthy hypnotics. They tend to depress the functions of the spinal cord, but their effect upon the heart, lungs, and other organs is small. Potassium bromide is the most depressing of the three, ammonium bromide the least; but in therapeutic doses this action is not very obvious, and the potassium salt is the most convenient to use. By many it is regarded as the most trustworthy.

Various other bromides also—for example, lithium bromide and hydrobromic acid—have been given as hypnotics; but none of these presents any material advantages over the salts more commonly used.

The bromides are useful in cases of insomnia due to worry or overwork. When relief cannot be obtained by non-medicinal means they are valuable to break the habit of sleeplessness, and thus to restore the brain to a more normal condition. In the presence of pain, however, they are generally useless. They are also the most useful drugs for repeated administration, as they appear to produce less serious ill effects than other remedies of this class. The dose may be given just before retiring, or, as recommended by Macfarlane, in two or three doses during the latter half of the day.

Chloral hydrate is a more powerful, but also a more harmful drug than the bromides. It produces general nervous depression, affecting both the brain and spinal cord; and it is also markedly depressant to the heart and vascular system. On this account it should be avoided in all cases of heart disease with symptoms of heart failure, and generally in diseases with a low blood-pressure, such as enteric fever. In cases of high arterial pressure, however, it may usually be given with impunity. Also in the more simple forms of insomnia, when bromides prove insufficient, to combine them with chloral hydrate is judicious and often successful. It should, however, be used with care, both on account of its circulatory effect and its tendency to form a habit. A suitable dose is 15 to 20 grains, or, combined with bromide, 10 to 15 grains; but sometimes more is required. Large doses should, however, be avoided.

Butyl-chloral will frequently relieve the insomnia due to neuralgia of the fifth nerve.

As substitutes for chloral, certain derivatives, purporting to possess its useful properties without its ill effects, have been recommended. Of these two only, chloralamide and chloralose, are of practical importance.

Chloralamide, or chloral-formamide, is undoubtedly less toxic than chloral hydrate, and does not produce the same depressant effect upon the circulation and respiration; but it is also a less powerful hypnotic. It may be used in doses of 30 to 40 grains in all cases where chloral is indicated, and in some forms of heart disease and bronchial affections.

Chloralose is a more powerful drug than either chloral hydrate or chloralamide. It differs from them in stimulating instead of depressing the functions of the spinal cord; in animals, to such a degree that convulsive tremors are produced. The heart is said to be uninfluenced by therapeutic doses; the blood-pressure is slightly raised. It may be used as a general hypnotic, and is especially recommended in cardiac and digestive troubles. In chronic Bright's disease, and others attended with increased arterial blood-pressure, it is contra-indicated on account of its influence in raising the pressure. Various opinions have been expressed about this drug. Some have found it beneficial, others not; and some have observed spasmodic twitchings from its use. It may be given in doses of from 3 to 6 grains in 1½ to 2 ounces of water a little while before going to bed.

Paraldehyde is a safer drug than any of the preceding. It produces sleep without any distinct respiratory or circulatory effect; but it is slightly irritant to the gastric mucous membrane, and possesses an unpleasant smell, which can be detected in the breath after its administration. On this account, indeed, it is not so likely to cause a habit, although a few cases of this are on record. It may be generally used as a hypnotic, and is specially serviceable in asthma, heart disease, and melancholia. It is best administered with tincture of orange, in a dose of ♏xl. to ♏xc., or upwards. Dr. Clouston has given ʒiv. to ʒvj. doses.

Sulphonal and *trional* are valuable hypnotics; the former in doses of 15 to 30 grains produces sleep in two to three hours, followed by no ill effects. Drowsiness is sometimes experienced during the following day; and the sleep of the succeeding night is often as sound, or sounder, than that of the night of administration. It possesses no action on the circulatory and respiratory systems. As it is slightly cumulative, repeated dosage may lead to hæmatoporphyrinuria and other untoward effects, and on this account it should not be continued longer than from four to five days. An interval of an equal or longer period should be allowed to elapse before resorting to it again, if this be necessary. Owing to the insolubility of the drug in cold water, it is best given in hot fluids, such as milk or soup; in this way a more rapid effect is obtained. Sulphonal is useful in all mild forms of insomnia, and one dose often produces two nights' sleep. It is adapted for continued administration, provided that intervals are allowed to elapse every few days, and that care be taken to avoid constipation.

Trional possesses similar properties to sulphonal, and exerts a similar effect. It is somewhat more powerful, and, being more soluble, it does not lead to a second night's rest, like sulphonal. Its uses are the same as those of the latter drug. It is valuable in the insomnia of children. Some authors say that it relieves pain as well as induces sleep.

Alcohol, in those unaccustomed to its use, is a valuable hypnotic in mild cases; especially in the form of beer, stout, or whisky, at bedtime.

I have ceased to use urethane as a hypnotic, as it is very uncertain in its action; and chloral-urethane or ural—introduced by Poppi—has no advantages over chloral, as in like manner it lowers blood-pressure.

Opium and its chief alkaloid *morphine* are the most useful hypnotics where there is great pain, and only in a few other cases should they be used; for instance, in recent and acute insomnia from worry, overwork, or shock. Their influence on the heart and vascular system is small, so that in heart disease, especially if attended with pain, they may generally be employed; but their influence upon the respiration is well marked, and in all respiratory diseases accompanied by impeded respiration from excessive secretion they should be regarded with disfavour. Opium inhibits most secretions, constipation and other undesirable effects usually accompanying its use. Rapid habituation also ensues, and in some patients marked excitement results from its first administration.

In painful maladies morphine hypodermically should be pushed until the pain is abolished; but on account of the well-known toxicity of the drug, care should be taken in its administration. In heart disease, $\frac{1}{8}$ to $\frac{1}{4}$ of a grain of morphine may be given subcutaneously, especially if there is stenocardial pain; or the drug may be taken by the mouth.

Opium is more slowly absorbed than morphine, and its action is correspondingly slower and more prolonged. It also possesses a greater local effect upon the alimentary canal, producing more marked constipation. It may be used to prolong the effect of morphine, or in cases, such as gastric cancer, where a local effect may be of value in aiding its general action.

Owing to the rapid establishment of tolerance, its ill effects, and tendency to induce a habit, morphine is not a good general hypnotic; and many attempts have been made to discover a substitute possessing its hypnotic and analgesic actions without its inconveniences, but so far without success. Certain derivatives of morphine have been suggested; but these are mainly sedative, and are not sufficiently powerful to be used in those cases where morphine is indicated.

Hyoscine, in the form of one of its salts, is one of the most powerful hypnotics we possess, and is said never to induce a habit. It is of special advantage in restless cases, such as the insane, where it is impossible to administer drugs by the mouth and undesirable to give opium. It is, indeed, the best substitute for morphine we possess; rarely producing excitement, and an atropine-like effect occurring only after considerable doses. Owing to its great toxicity care must be taken in its administration; $\frac{1}{200}$ to $\frac{1}{150}$ grain of the hydrobromide, given hypodermically, is usually sufficient, under ordinary conditions, to produce sleep; but in the insane large doses—$\frac{1}{100}$ grain, or even $\frac{1}{50}$ grain—are often required. These quantities should not, however, be given as initial doses. Valvular disease, so common in acute mania, is a contra-indication to its use. A combination of chloral with $\frac{1}{120}$ grain of hyoscine, or with morphine, has given excellent results in cases of obstinate insomnia with excitement.

Hyoscyamine has been used as a hypnotic in cases in which hyoscine is of benefit, but it is not so valuable. It produces more numerous ill effects, and its sedative action is less powerful. The other members of the atropine group are even less useful. Hurd recommends a teaspoonful of the tincture of hyoscyamus at bedtime as a useful hypnotic in cystitis.

Cannabis indica is used by many as a hypnotic. It is, however, apt to produce excitement, and sometimes this is not followed by sleep. It does not produce constipation as opium does, and it is less toxic; in fact no fatal case of poisoning by this drug is known. Its greatest drawback is its inconstant composition owing to its deterioration with age, and so far no official action has been taken to obviate this. Several preparations are on the market. The best are the extractum cannabis indicæ of the British Pharmacopœia, Merck's cannabinon and extractum cannabis indicæ, and cannabinol. A convenient dose to begin with is half a grain of the extract, but with many samples this amount has to be exceeded. There are no special indications for its use. It is given in migraine and neuralgia, and often alleviates the pain of gastralgia in old people; but it is not a powerful analgesic. In other forms of pain it is less valuable. It may be regarded as a general hypnotic except in cases where its excitant action is contra-indicated; and if a good preparation can be obtained, it appears to be a valuable one. It is an ingredient of the much-advertised hypnotic "bromidia."

Pellotine, an alkaloid of a species of cactus (Anhalonium Williamsii), has been recently advocated as a hypnotic. Sufficient clinical data, however, have not yet been obtained to establish its value; and, as far as we can judge, the results of purely scientific investigation do not prove it to be better than other well-known drugs. It has been used in insomnia from various causes. In locomotor ataxy and other painful conditions sleep was produced in some cases, but the pains returned on awaking. No unpleasant after-effects are said to occur, but giddiness, restlessness, and slowing of the pulse are occasionally noted before sleep takes place. The dose recommended is ¼ to ⅓ of a grain, but Jolly states that 1 grain is the proper dose.

Two other agents may be used to induce sleep if the measures previously considered fail; namely, electricity and hypnotism.

Electricity.—Electricity is regarded as a valuable agent by those accustomed to use it, but it is generally recognised that its employment is empirical. In some cases it produces excitement rather than sleep, and it is said "to be largely a matter of experiment as to who will and who will not sleep better after its use." In the majority of cases, however, sleepiness is induced, and, according to Eskridge, the cases most commonly benefited are those in which the insomnia is due "to mental overwork, worry, alcoholism, and supposed hyperæmic conditions of the brain generally." Riggs also states that it is most useful in the sleeplessness accompanying nervous and mental disorders.

Various modes of electrification—general faradisation, galvanisation of the head, cervical sympathetic, etc., and static electrification—are in use. Dr. Lewis Jones recommends general faradisation or the faradic bath, but the majority of electricians are in favour of galvanism. Weak currents (1-3 milliampères) directed longitudinally and continued until the patient feels drowsy, sometimes 15 to 20 minutes, or even 30 minutes, and repeated on alternate days, appear to produce the best results.

In some cases static insulation is preferred, and positive insulation is said to be better than negative (Rockwell). The method as practised by Dr. H. McClure, late of Cromer, is as follows: the patient, insulated on a glass stool, is brought into connection with the conductor of a Carré or Holtz machine. When thoroughly electrified a fine metal point is held opposite several spots on the scalp and forehead, but not near enough to produce a spark. The sensation is as if a light wind or breeze was playing over the part, and sleep is said often to come on while the treatment is being employed (Whitla).

The influence of electrification, in whatever way it is used, is said to be both temporary and permanent. The temporary effect occurs during the first few nights, and is never so great as that obtained from drugs. But the permanent influence is the end to be obtained. As compared with drugs electricity possesses the advantages of being free from ill effects, and the danger of forming a habit, but in some cases it fails to act, and may even aggravate the symptoms; consequently its effects must be carefully watched (Riggs).

Hypnotism.—Cases of insomnia, rebellious to all other forms of treatment, may be treated by hypnotic influence, but only in the hands of a properly qualified and specially experienced medical man. Under such conditions I believe it to be a valuable therapeutic agent.

SOMNOLENCE.—Somnolence, or excessive sleepiness, as previously mentioned, occurs in persons of feeble intellect. Some old people also sleep a great deal. In cases of organic brain mischief, especially cerebral syphilis (Buzzard), and in some epileptic and hysterical persons, somnolence is met with (*vide* art. "Hysteria"). It has also been attributed to the reflex irritation caused by round worms, as the removal of these has caused the somnolence to cease. Some sufferers from anæmia, leukæmia, and myxœdema sleep too much, the last more especially in the daytime.

The sleeping sickness, or negro lethargy of the West Coast of Africa, is, as its name implies, attended among other symptoms by drowsiness, which gradually deepens into somnolence and profound coma (see article on this disease, vol. ii. p. 479).

DREAMS AND NIGHTMARE occur when certain cortical centres are active, whilst others are in abeyance. They frequently arise from indigestion; but occasionally they are the forerunners of apoplexy or insanity. "The recurrence of a peculiar and unusual dream night after night, particularly if it is of a terrifying kind, should always arouse suspicion" (Macfarlane). A patient of mine, an old lady of eighty-one, had a most distressing dream a day or two before an attack of right hemiplegia.

The NIGHT TERRORS of children arise in connection with alarming dreams. They usually occur in neurotic children, from one to four years old, during the first hour or two after they go to bed, when the sleep is most profound. Dyspepsia is the commonest exciting cause. Children troubled with this affection need soothing by a mother or skilful nurse, careful dieting, a stomachic mixture or powder, and a dose of bromide at bedtime for a few nights in succession to calm the cerebral centres of emotion.

SLEEP-TALKING or somniloquy, and SLEEP-WALKING or somnambulism, are states in which the whole brain is not asleep, but certain centres continue in activity. Some persons, whilst apparently asleep, may even carry on a conversation, as if they were awake.

Somnambulism, which has been described as an acted dream, runs in families, and occurs about equally often in the two sexes. The subjects of it can perform extraordinary feats, such as unlocking doors, walking in dangerous places, as on the edge of a precipice in the dark, scaling a partition of considerable height (8 to 10 feet) between two sleeping-apartments; and they usually do all this without injuring themselves.

It is necessary in some of the worst cases to keep the patient in a room locked from the outside, to fasten all windows, and occasionally to chain one leg to the bed, so as to wake him if he begins to walk. The general health should, of course, be carefully attended to.

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EPILEPSY

The Name.—Epilepsy is a disease of which the chief feature is the recurrence of sudden brief attacks in which consciousness is lost, and with it the control of the muscles. In consequence the patient falls, and the malady received from our ancestors the apt symptomatic name of the "falling-evil" or "falling-sickness." But the muscles commonly pass into a state of involuntary contraction, sometimes slight, but generally intensely energetic, fixing the frame in some strained posture, stopping the breath until the aspect is that of strangulation, and passing away in violent shock-like spasms which leave him in a state of unconsciousness with life only not extinguished. To the Greeks it seemed that the attack must be the work of some unseen agent of demoniacal ferocity and strength, and hence they gave the disease the name of epilepsia, a "falling upon," as by a spiritual agent. This involved a theoretical pathology which was apparently absent from the simpler Anglo-Saxon conception. The same idea, however, is preserved to us in the popular designation "a seizure," and is not quite absent from the name applied so widely in the description of diseases as "an attack."

Definition.—More careful observation has shown that neither fall nor convulsion is a predominant or even dominant characteristic of the disease, common as each feature is. It has shown also that the attacks which constitute the malady present variations so great, both in degree and in character, as to baffle every attempt to frame a clinical definition. The only definition which embraces all forms is "recurring attacks, sudden and very brief, of disturbances of some of the cerebral functions, acting on consciousness, which are not due to a cause outside the brain." But this definition is at once too wide and too narrow. It would exclude ases in which local spasm continues for hours, and it would include nany cases which must be classed as pure vertigo and not as epiepsy, while it would not include the cases in which convulsions are he result of a local injury to a nerve, cases which are, it is true, rare nough.

A nearer approach to adequacy may be found in the pathological lefinition that epilepsy is recurring sudden brief discharge of nerve nergy in some part of the cerebral cortex, not due to the normal cause f such discharge. Exceptions may be taken even to this description, nd their character will appear in the sequel. A definition of epilepsy vould not indeed be worth even this slight attempt, were it not that the ailure may make some facts, to be mentioned presently, more instructive. The only use of defining is to enable us to see better what is on each side f the line. But Nature, when she can, defines a country by a winding iver or a range of mountains. We like to ignore all this, and draw traight lines.

Pathological definition.—Accordingly it may be worth while to see vhat the pathological definition marks off. The sudden discharge of ierve force, so conspicuous in a convulsion by the muscular spasm it roduces, may be traced also in all the minor forms of the attack. In he sensory centres it acts, as we say, "upwards," so as to influence onsciousness only, and may cause, for instance, an appearance of bright tars. When the attack consists of loss of sight, or simply of loss of onsciousness, there is an apparent exception. We associate "discharge" vith over-action. But over-action in one part may arrest action in nother.

The sudden liberation of nerve energy in the gray matter of the erebral cortex occurs in epilepsy without the normal stimulus. There s much in this statement that will render the facts of the disease more ignificant, although it is only possible to glance very briefly at that which underlies it.

The function of the nervous system, and especially of the cortex of the brain, depends upon a capacity for instant release of nerve energy. The instantaneous reflex action, the instant bound away of a frightened animal, prove that the gray matter must hold nerve force almost released. Released from what? We must regard the energy of nerve and muscle which is excited by a touch upon the skin as a form of physical energy, peculiar because acting under the influence of life, released and conducted in living tissues. It is probable that the energy from which nerve force is released is latent chemical energy; with this latent chemical energy is inter-atomic and inter-molecular motion, and that nerve energy is a form of transmitted motion by transmitted chemical processes. The reasons for thinking this I have stated elsewhere (1).

The point for us now is that the nerve force must be ready for instant release in the gray matter. Whatever its source, it must be held in a state of most delicate equilibrium by the restraint which keeps it unreleased but ready. Indeed, there is good reason to think that, to use a simile, the vessels which hold it must be not only filled to the brim and delicately poised, but must be actually overflowing. Both the facts of the constant process of nutritional change, and the evidence of constant activity in the nervous system, lead to the conviction that the readiness of the nerve energy is obtained by its constant overflow, that the chemical processes which induce its sudden generation are always going on in slight degree, and that only thus is it kept so as to be released at once in the needful degree.

But this involves a conception of extreme *delicacy of adjustment* between the chemical relations—the tendency of the atoms to unite in fresh and closer combinations and release their latent energy, that is, their surplus motion—and the attraction which keeps them from so doing. It makes it easy to conceive that the slightest deviation from the normal relation between the atoms and the molecules may bring these into a position in which the equilibrium is suddenly overturned, and a quick combination and sudden release of energy occurs. We can understand that this may be from that marvellous inherited tendency which produces such precision of correspondence between parent and child in the process of nutrition, or that it may be the result of acquired causes, from the sudden shock of a concussion, mental or physical, or of a cerebral lesion which has slightly, but only slightly, implicated the gray matter in a certain part of the cortex. Indeed, when we consider what the state of the nervous system must be in its natural function, we cease to wonder that many persons become epileptic without any traceable cause, and our marvel rather is that all persons are not liable to such fits.

The release is normally effected by a stimulus of a certain character acting in a certain direction among the complex molecules of the nervous system, the arrangement of which facilitates its transmission and action. In the lecture referred to (4) it is maintained that such a stimulus may be best regarded as atomic motion which, added to the pre-existing motion of the latent energy, causes this to form the new and fresh combinations in which there is less constant motion.

A morbid relation of the most delicate and minute character between the molecules may make them susceptible to motion which comes by some other path which we do not discern. Discharge without the normal stimulus may not mean discharge without any stimulus: yet even this is possible; we can conceive that the defective equilibrium may entail a disturbance of the balance, when a certain point is reached, and a discharge which has no other cause than the nutritional augmentation of its own tendency.

The important fact thus is that an extremely slight derangement of the constitution of the nerve tissues may cause a liability to sudden discharges of nerve force, slight and local or severe and wide.

Recurring tendency constitutes the disease.—But another equally important fact must be recognised. The liberation of nervous energy involves the escape of some atoms of the nerve tissue which are at once replaced from the plasma adjacent to them under the mysterious nutritional influence of life. The rearrangement is the same, and yet not quite the same. It differs in just the degree and character to make the same release of nerve energy more easy. Every action paves the way for its own recurrence. The effect is greater the more frequently its occurrence. This is the secret of the acquisition of skill; it constitutes the physical basis of memory; it is this which facilitates the repetition of a given morbid process, and this causes the recurrence of discharge which makes epilepsy a persistent disease. The recurrence of attacks is one great feature of epilepsy, and the recurrence is in great measure the result of the previous attacks. Every fit is in part, at least, the result of those which have preceded it, and in part a cause of those which follow it. Hence the immediate cause of the first fit must be regarded as the cause of the disease, although the essential cause is the disposition, that is, the state of nutrition, which makes such discharge possible. When equilibrium is unstable a trifling influence may overturn the balance, and cause an effect quite out of proportion to the apparent cause. The degree of instability may, of course, vary; and when it is not great the first attack may have been due to some cause of considerable power. Even then, however, we must ascribe the special effect to the special predisposition.

These considerations will make it easier to understand the causes of epilepsy in so far as they are comprehended. But one other important question must also be noticed.

General and local change.—In what is called "idiopathic" epilepsy, that which is largely the result of inheritance, the morbid state consists in some trifling alteration in the chemical constitution of the gray matter in which the instability spoken of consists. But a similar instability may be the result of obvious disease. It is not indeed directly due to disease that can be seen in the damaged part, because for disease to be visible the destruction of nerve tissue must have made even morbid action impossible. But beyond the visible disease, whatever its nature, is a zone of altered nerve tissue in which the slightness of the change permits activity in altered form. In such cases the discharges spoken of may take place. Arising locally, they spread through the brain according to their energy, and then leave a tendency to repetition, which involves parts unaffected by the primary disease. It is not customary to include under the term "epilepsy" active progressive brain disease, such as a tumour, even though it cause epileptiform convulsions. But there is a large class of cases in which an old spot of disease induces such discharges, which may be slight or local at first, but spreading when more intense, and ultimately leaving a general disposition which may be hardly distinguishable from idiopathic epilepsy. Although these cases are quite distinct in primary causation, they are not practically separable from those of idiopathic epilepsy.

Perpetuation of reflex convulsions.—A third group occupies a curiously intermediate position. There are cases in which some local irritation, for instance in the intestinal canal—generally tapeworm—has excited "reflex convulsions," which had been so many times repeated before their cause was removed, that a persistent habit of brain function was established. This may happen even without any inherited disposition. It is possible that some other influences, acting on the nervous system from the outside, may have a similar result. Such cases do not ultimately differ in any important way from the common idiopathic disease, but it is necessary that their initial character should be discerned.

Source of the discharge.—I have spoken of the discharge of nerve energy as proceeding from the gray matter and not from the nerve-cells; and it may be noticed that nothing has been said of morbid changes associated with the idiopathic disease. The two omissions are connected in a significant manner. Up to the present time it has been a fundamental conception that the nerve-cells from which the fibres proceed are the source of the nerve energy which passes along the fibres that proceed from them. Apart from any opinion regarding the electrical nature of nerve force, the obvious analogy between the large cell as a generator and the sheathed fibre as a conductor, has aided in fixing firmly the conception. But the labours of histologists, of Max Schultze thirty years ago, and of Ramon y Cajal, of Schäfer, and others in our own day, coupled with the known facts of physiology, make it necessary to relinquish this simple and attractive notion. We must, it would seem, regard the function of the nerve-cells as nutritional only. On them depends the life both of the processes, short or long, which conduct to them, and the long axis-cylinder process which conducts from them. Through the cell pass the separate fibrils of which the apparently homogeneous axis-cylinder really consists. These arise, without continuity with others, in the spongy gray matter; becoming associated in the "branching processes" they reach the nerve cell to pass through its protoplasm without interruption. We must regard the spongy gray matter as the substance in which the nerve impulses arise under the influence of other impulses conducted to it; and we must therefore look to this, and to this alone, as the place in which any alteration exists that underlies the phenomena of epilepsy. The eyes of the observer hitherto have been fixed on the cells and on the cells alone. We do not yet know how to unravel the complex structure of the spongy substance so as to discern the normal relations and aspect of its constituents. We are not yet on the threshold of the observation of such local disease as must exist in idiopathic epilepsy. Perhaps we never shall be. But it is at least easy to see why we at present know nothing of the pathological anatomy of the malady. Even the aggregations of atoms which form the organic "molecules," under the influence of life, are far beyond our power of observation; so far, indeed, that a vast amount of change, involving extensive and constant functional disturbance, may result from a change in nutrition which cannot be recognised by any means at our disposal. Any perceptible alterations are more likely to be the result of the functional disturbance than the cause of it. Functional action involves local vascular disturbance, and this, if intense, may be manifested by such visible effects as are seen in the medulla oblongata in hydrophobia. But before we can hope to discern that which is connected with the finer disease of the spongy gray matter, we must first become conversant with its minute features under normal conditions.

Causes.—The preceding sketch of the etiology of epilepsy should make it easier to understand the facts of its causation, and should also make it unnecessary to dwell on their significance. The most important impression which the reader should retain from it is that the conditions of the normal action of the brain involve a state of tension ready for release of energy, and a delicate equilibrium which makes the abnormal sudden liberation of energy readily produced, either in consequence of a slight primary perversion of the conditions on which the balance depends, or a sudden and excessive shock to them.

Heredity.—Epilepsy is an inherited disease. Epilepsy and insanity certainly run together in families. The general tendency to one involves also the tendency to the other, although not often in the same individual. In seeking for the family antecedents of epilepsy, it is found that insanity is the only other disease to be taken into consideration. Other neuroses have too many different connections, or these are of so common a kind as to be comparatively insignificant. But if only epilepsy and insanity are considered it is found that in half the cases of epilepsy there is a family history of one or both of these diseases. If the facts be gathered from the lower classes, who seek relief and yield data at the hospitals, this association can only be traced in a third of such patients. But when cases are carefully investigated in private, the proportion rises to one-half : and even this estimate is doubtless below the truth. In the present generation the morbid facts are generally known, but unpleasant events in antecedent generations are often unmentioned in a family, and the knowledge of them may thus be lost.

Of diseases outside the nervous system, it is not possible at present to discern any which stand in a definite relation to epilepsy. Phthisis was once thought to be connected with the disease by some hereditary mechanism, and the cases of phthisis to be heard of in the families of epileptics are many. But the ratio is not found to be larger than in the families of those who are not epileptics, and an investigation from the side of phthisis clearly showed that the apparent connection was due to the frequency of phthisis. In the same manner it is not possible to say whether inherited gout has an influence in the production of epilepsy. It is difficult to resist the evidence of a connection between inherited gout, migraine, and neuralgia; but the connection between these diseases and epilepsy, although definite (at least so far as migraine is concerned), is yet so slight and infrequent as rather to oppose than support the idea that inherited gout is a factor in the production of epilepsy. That the individual subject of epilepsy may suffer more in consequence of the systemic disturbance of gout is of course intelligible without any causal relation.

The consanguinity of parents necessarily intensifies the family habit, but there is no evidence that it has any influence in producing the disease.

Such marriages are indeed far too rare to furnish any material from which an opinion can be formed.

Sex.—The influence of sex at all ages is not great. For every twenty males twenty-one females suffer from epilepsy. This is the result of a careful comparison of nearly 3000 cases, from which all examples of hysteria, not associated with true epilepsy, have been excluded. In some small collections previously published males have slightly preponderated, possibly from the inclusion of cases of syphilitic brain disease.

Age has a great influence on the production of epilepsy. This may indeed be expected from the tendency to spasm and convulsion which is conspicuous in childhood, which lessens as the stability of full development is achieved, and is again increased during the change with which the period of childhood ends. The periods of this change, and of the first three years of life, are those in which epilepsy most frequently commences. One-eighth of all cases begin in the first three years of life, transient infantile convulsions being excluded. Not less than a quarter of the cases begin in the four years, 13, 14, 15, and 16, which, in the majority of cases, include the epoch of puberty. In the second decade, indeed, the malady commences as a continuous disease, in no less than almost half the cases; and in the first decade in more than a quarter: thus three-quarters of the cases of epilepsy begin before 20 years of age. The third decade is responsible for another sixth, leaving only one-twelfth to commence after 30 years of age. But the persistently recurring fits may begin as late as 70 years of age. Yet only one case in 200 commences after 60.

Sex and age.—There is apparently an absolute identity of the two sexes in the early period of embryonal development. There is a similar correspondence between them in their nervous characteristics in the early period of separate existence, especially during the first two or three years of life. Yet the existence of a profound difference in the nervous system of the two sexes is shown by the fact that in cases of epilepsy commencing in the first ten years of life the females exceed the males by 8 per cent, and the difference is greatest in the first year, in which the males are little more than one-half the females. Puberty has a far more profound influence on the nervous system of females than on that of males, and their excess rises, in the second ten years of life, to 20 per cent. With adult life the influence of sex is changed. In the third decennium the disease begins in the same number of males and females. Between 30 and 40 the males are for the first time in excess by no less than 20 per cent. Between 40 and 50 their excess attains nearly 30 per cent. After 50 the same relation persists, but the numbers are too few to permit accurate comparison. Cases of idiopathic epilepsy begin in males up to 75, but almost, if not quite, exclusively in males. We may find an explanation of these facts in the exposure of the nervous system to more constant strain of anxiety in men, but it may also be that some compensatory indisposition is developed in women who have maintained stability through the trying period of development. Certainly facts yield

It is therefore thought by many that Weichselbaum's D. intracellularis meningitidis is the true cause of the epidemic form of cerebro-spinal meningitis.

The bacteriology of posterior-basic meningitis, of the type described in this paper, has been investigated by our friend and colleague Dr. Still, Medical Registrar and Pathologist to the Hospital for Sick Children, who has kindly given us the following report:—

"*The bacteriology of posterior-basic meningitis.*—The presence of a diplococcus in pure growth in the exudation at the base of the brain has now been demonstrated repeatedly in the simple posterior-basic meningitis of infants. There is good reason for supposing that the disease is due in all cases to the presence of this micro-organism. Examination of nine consecutive cases showed that the diplococcus was present in all except one case, which proved sterile. The sterility in this case was probably due to the fact that death occurred nearly four months after the onset of the disease, when all exudation had already been replaced by fibrous adhesions. It seems probable that in such cases the micro-organism may have disappeared completely; but subsequent experience has shown that even after the lapse of nearly four months from the onset the micro-organism may still be found in the cerebro-spinal fluid in the lateral ventricles.

"The diplococcus of posterior-basic meningitis consists of cocci which vary in shape from an almost complete sphere to a hemisphere, and have their opposed surfaces flattened or even concave. The two cocci can be seen to be separated by a narrow clear space. It is smaller than the pneumococcus and does not show the lanceolate form so common in pneumococcus. No definite capsule has yet been demonstrated. Both in the exudation at the base of the brain and in cultures there is a tendency to grouping in pairs, so that an appearance like that of M. tetragenus, or of sarcinæ, is produced. In growths on solid media two or even three diplococci are occasionally seen joined end to end, but this arrangement is rare, and there is no streptococcic growth like that seen in cultures of pneumococcus. In the exudation the diplococci sometimes appear to be intracellular, but such an appearance is exceptional, and in most cases is not seen at all. There is no difficulty in staining it by any of the ordinary methods; save that this diplococcus does not stain by Gram's method—an important means of distinction from the pneumococcus. It is aerobic, and grows in the incubator at a temperature of about 37° C.; no growth occurs at the ordinary temperature of the room. Agar-agar or glycerin-agar may be used as the medium; on either of these growth is rapid, and in subcultures may be recognisable within four hours. If some of the exudation from the brain be smeared on these media the growth is seen as small, round, slightly raised colonies of a grayish white colour which tend to run together to form larger irregular rosette-shaped patches; in streak subcultures the growth consists of a slightly raised grayish white streak with a tendency to beading at the margin. In either case the thickness of the growth and the larger size of the colonies contrast markedly with the very thin, translucent, minute colonies of the pneumococcus. On blood-agar—that is, agar on the surface of which there is a thin layer of sterilised blood—growth is very rapid and very profuse; it has the appearance of thick white paint, and contrasts with the smaller yellowish colonies of the pneumococcus on this medium. On blood-serum growth is very slow and extremely scanty. There is no growth on potato, nor on gelatin at the ordinary temperature of the room. In broth this diplococcus grows well, producing at first cloudiness of the fluid, and subsequently a filmy deposit at the bottom of the tube; herein it differs from D. intracellularis, which is said by Weichselbaum to show 'almost no growth' in broth. In milk growth is abundant, but no coagulation is produced—a further point of distinction from the pneumococcus, which certainly often, if not usually, produces coagulation. The addition of a trace of sulphuric or lactic acid to agar-agar or glycerin-agar prevents the growth of the diplococcus on these media.

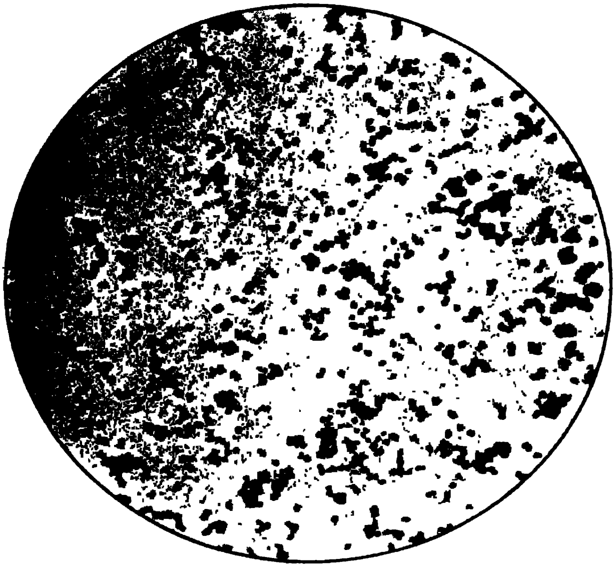
FIG. 88.—The diplococcus of posterior-basic meningitis. From a photograph. Thirty-one hours' growth on blood-agar. (×1000.)

"The most striking difference between this micro-organism and D. intracellularis is the much greater vitality of the former. Weichselbaum lays stress upon the remarkably short vitality of D. intracellularis as one of its chief characteristics, and states that in no case did it live beyond the sixth day; whereas the diplococcus of posterior-basic meningitis shows in almost all cases a maximum vitality of twenty-four to thirty-four days on ordinary media (agar-agar and glycerin-agar), and on blood-agar lives considerably longer (fifty-three days in one case).

"The diplococcus of posterior-basic meningitis is pathogenetic for guinea-pigs, rabbits, and mice, when inoculated either by intraperitoneal or (in the case of rabbits) by intravenous injection. Subcutaneous inoculations produce little or no effect—a further important distinction from the pneumococcus. The variability of results of intraperitoneal and intravenous injection is, however, very marked, and in this respect this diplococcus seems to differ from the D. intracellularis, which would seem to have a more constant virulence.

"The etiological relation of the diplococcus here described to the posterior-basic meningitis of infants seems to be further confirmed by the discovery of the same micro-organism in the periarthritis which occasionally complicates this disease. The similarity of the exudation about the joints to that in the meninges had already suggested identity of cause, and it has now been shown (18) that the diplococcus of posterior-basic meningitis may be found in pure growth in the exudation about the capsule of the joint and the neighbouring tendon-sheaths in these cases.

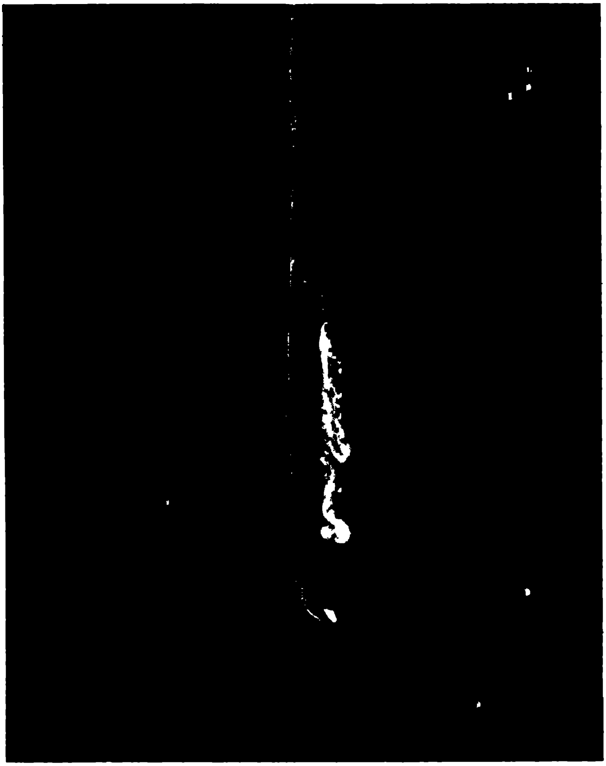
FIG. 34.—The diplococcus of posterior-basic meningitis. Growth on agar-agar.

"It will be seen from the account given above that while the diplococcus of posterior-basic meningitis resembles in many points D. intracellularis as described by Weichselbaum, it shows also certain constant differences. It seems most probable that these differences are the result of natural variation, and represent rather a modification of characteristics than a distinction of kind."

Duration.—The average duration of the 14 "vertical" cases was 8·7 days, the limits being two days and twenty-four days, the latter limit being a little uncertain. Vertical meningitis is therefore a disease of short duration; it is more rapidly fatal than tuberculous meningitis.

The average duration of 30 fatal cases of posterior-basic meningitis was 7·8 weeks, the limits being three weeks and fifteen weeks. But some cases are of still longer duration, and when hydrocephalus supervenes it may be impossible to determine precisely the duration of the meningitis. Two cases not included in the 30 lasted six months and ten months respectively. Another lasted for 2½ months, the child apparently recovering; but death occurred unexpectedly seven months later, when hydrocephalus and obliteration of the fourth ventricle were found. Another patient apparently recovered in thirteen weeks, but, three months later, died after twenty-four hours' illness and was then found to have hydrocephalus and adhesions between the cerebellum and medulla obliterating the cerebro-spinal and lateral foramina. Another recovered in four months, but its head was then too large; it died two years later after two days' illness; there was no necropsy. Thus the average duration of fatal posterior-basic meningitis is more than twice as long as that of tuberculous meningitis.

It seems impossible to state an average duration for cases that end in recovery. Some are slight from the first (though it must not be forgotten that very slight cases may be followed by hydrocephalus and mental defect). A few are distinctly arrested by treatment. Recovery is so gradual in a severe case that it may be very difficult to fix the time when the child may be considered well. Many pass from observation before recovery is complete.

Of our 94 cases of posterior-basic meningitis, 13 ended in complete recovery; and 2 others probably did so, giving a total of 15 complete recoveries, or a proportion of 1 in 6. The fatal cases, on the other hand, numbered 45, or 1 in 2.

Recovery is often incomplete. Some cases are obviously hydrocephalic; the amount of distension of the head, as a rule, varying inversely as the age when the obstruction is established. Some patients remain blind (with no optic neuritis) for a considerable time at least; in one case the child was still blind eight months after the onset, in another eleven months afterwards; recovery in other respects also being imperfect. Others become deaf, and some of these certainly remain permanently deaf, and as a consequence gradually lose the power of speech, if they have previously acquired it. Many are backward in learning to stand and to walk. Some are not clean in their habits. Many are obviously more or less defective in intelligence. It is not uncommon for children who have thus imperfectly recovered to die unexpectedly after a very brief illness.

Diagnosis in meningitis is sometimes difficult. An illness of acute onset, marked by rapid rise of temperature, vomiting, headache, and convulsions, may be meningitis, especially of the vertical kind; but it may be simply the onset of scarlet fever or of diphtheria; or it may be a pneumonia in which the cerebral symptoms are more obtrusive than the pulmonary; or again it may be a severe anterior poliomyelitis. A day or two later a sore throat, an eruption, signs of consolidation of lung, or local paralysis may decide the diagnosis. The most deceptive of these conditions is pneumonia; for it may be difficult to detect physical signs in the lungs for several days, and meantime the cerebral symptoms may be pronounced. Special attention should be paid to the frequency of respiration, and a rapid respiration-rate should lead to a very careful examination of the lungs. The dilatation of the nostrils at each inspiration will often indicate the real seat of disease. But the recurrence of convulsions or vomiting should direct special attention to the brain, though the absence of convulsions must not be considered decisive.

If the onset of the illness be somewhat less acute, it will be necessary to consider whether the illness may be typhoid fever, which sometimes has rather an abrupt onset. Acute miliary tuberculosis also, which sometimes resembles typhoid very closely, must not be forgotten.

If the head becomes strongly retracted within the first few days, the cause is probably either otitis or posterior-basic meningitis. In otitis there may be some lateral inclination of the head combined with the retraction; the child may raise its hand to the side of its head, and it may be clear to a careful observation that there is pain in one ear. If there be no symptom of this kind, and the head-retraction become very marked and persistent, the diagnosis of posterior-basic meningitis is almost certain.

If the retraction of the head be slight, amounting to little more than stiffness of the neck, vertical or tuberculous meningitis is possible; but it may be due to pneumonia. Head-retraction which is variable or transitory is rarely due to posterior-basic meningitis.

Cases of traumatic origin are rarely of tuberculous nature. Whether the vertex or the posterior-base be mainly affected will be determined by the locality of the injury, or possibly by contre-coup. Head-retraction preceded by catarrh is almost always posterior-basic.

The age of the patient is of importance in the diagnosis. Simple meningitis is much more common than tuberculous during the first year of life, and about equally common during the second year; but at ages above two years meningitis is most frequently tuberculous.

Convulsions and localised clonic spasms are usually of cortical origin, but there may be some irritation of the cortex in posterior-basic meningitis; on the other hand, the cortical cells may be so overwhelmed by a suppurative generalised meningitis that there may be no convulsion at all.

No weight whatever must be attached to the absence of otorrhœa in the diagnosis of otitis or of posterior-basic meningitis.

Screaming at the onset and marked occipital tenderness point to posterior-basic meningitis.

The longer the duration of the disease, the less probable is suppurative vertical meningitis. If the illness have already lasted from two to three weeks, and the head-retraction is slight and variable, the meningitis may be vertical or tuberculous, but it is less likely to be posterior-basic. But if the head-retraction is decided, and has been persistent for a fortnight, the case is almost certainly posterior-basic. We have seen a few cases in which tuberculous meningitis produced so marked and persistent a retraction of the head as to lead to an error in diagnosis; in these a greater development of tubercle than usual was found in the region of the cerebellum and medulla. Such cases are very rare. A meningitis which has lasted more than four weeks is more likely to be posterior-basic than either vertical or tuberculous.

The condition of the pupils is of no value in the differential diagnosis; and the same may be said of strabismus. But nystagmus is much more common in the posterior-basic form than in the tuberculous, and we have not seen it in the vertical. Distinct optic neuritis is strongly in favour of the tuberculous nature of meningitis, though it is not unknown in the two other forms; but when optic neuritis is of high degree, the possibility of the existence of a cerebral tumour ought to be considered. Amaurosis in a child not comatose, with absence of optic neuritis or atrophy, is very characteristic of posterior-basic meningitis, and may persist when other symptoms of the disease have vanished.

The respiration may be irregular and sighing in all forms of meningitis, and in all it may assume the Cheyne-Stokes rhythm. But the type of breathing which we have called "cyclical," in which the respirations in each cycle are of equal depth, occurs specially in the posterior-basic form.

Slowness of pulse is less common in posterior-basic than in tuberculous meningitis, probably because the former is the more common in infancy, the symptom being rarely met with under twelve months of age.

Marked retraction of the abdomen is strongly in favour of tuberculous meningitis, though it may be present in less degree in the posterior-basic form.

Persistent tonic spasm, leading to rigidity of the limbs, is characteristic of posterior-basic meningitis. Some rigidity of limbs may occur in the tuberculous affection, but it is much less persistent than in the posterior-basic type; and extreme rigidity of the limbs and marked opisthotonos are confined to this type.

Bulging of the fontanelle and slight separation of the sutures may occur in any form of meningitis in infancy; but decided and progressive enlargement of the head, as a sequel of meningitis, is found only in the posterior-basic (occlusive) type.

Prognosis in vertical meningitis is only a little better than in tuberculous meningitis. Cases following injury are more hopeful than the idiopathic cases, for in the former there is less likelihood of extensive suppuration.

The prognosis of posterior-basic meningitis is decidedly more favourable than that of the tuberculous affection. This is true for infants, but it *is still more true when the disease occurs in children of more than a year old or in adults, for in these the prognosis is better than in infants.* If the symptoms are slight, or if in a severe case vigorous treatment is adopted at an early stage of the disease, recovery is by no means hopeless. Unfortunately many cases are not brought for treatment until irreparable mischief has been done. In some of our fatal cases the condition found on necropsy was such that recovery might have occurred but for some cause wholly or partly independent of the meningitis, such as diarrhœa or exhaustion. But it is important to remember that subsidence of the cerebral inflammation does not necessarily mean a final recovery, and a very guarded prognosis must still be given; for even after apparent recovery hydrocephalus may gradually ensue, and weeks or months later may prove fatal. Careful measurements of the size of the head should be recorded weekly, and the subsequent development of the cerebral functions closely watched.

Amaurosis generally disappears if life is prolonged. Permanent deafness is rare, certainly more rare than in the epidemic disease—perhaps in consequence of the less degree of virulence of the micro-organism. But when deafness is permanent it may cause loss or arrest of speech, so that the child becomes a deaf-mute. A considerable degree of mental defect, even amounting to imbecility, is not uncommon.

Finally, it should be remembered that a case in which a fair recovery seems attained, may suddenly and unexpectedly end in death, when hydrocephalus may be discovered.

Treatment.—The first point for consideration, in cases seen at an early stage of the disease, is whether paracentesis of the tympanic membranes should be performed. This operation has definitely arrested the symptoms in several cases under our care. Thus a child, aged 18 months, whose head had been retracted for three weeks (after an attack of bronchitis), with frequent vomiting, much irritability, nystagmus, occasional twitching of right hand, and tendency to raise its hand to its left ear, was sent to Mr. Field, aural surgeon to St. Mary's Hospital, who punctured both tympanic membranes, and let out pus from the left ear. The child seemed much relieved immediately after the operation. Next day it was nearly well, and in five days the head-retraction had quite disappeared.

Another child, aged 16 months, had had marked retraction of the head for seventeen days, with occasional discharge from the left ear, and at first prominence of the fontanelle. Paracentesis of both tympanic membranes was performed by Mr. Field. No pus was obtained, but three days later there was a spontaneous discharge from the right ear, and the next day the retraction had nearly disappeared; a little remained for about five days.

A third infant, 6 months old (case published in the *Practitioner* (10) for 1886), after a severe attack of broncho-pneumonia, had moderate retraction of the head for two days, with some divergence of eyes, contracted pupils, tense fontanelle, slight jerks of forearms and hands, partial coma, occasional sighing, and occasional flushing. Mr. Field punctured both tympanic membranes; a little blood came from both, but no visible pus. Improvement followed at once, and in two or three days all the above symptoms had vanished. Ten days later she had obvious pain in the left ear, and fourteen days after the operation she was again in a semi-comatose state, with diverging eyes, with marked and frequent spasmodic contractions of the facial muscles, and with some jerks of the hands. Paracentesis of both tympanic membranes was repeated. On this occasion distinct pus came from the left ear, a little blood from the right. The spasmodic movements ceased at once, and recovery followed. This child is now a well-developed girl of thirteen years, with good intelligence and normal hearing.

In all these cases, and in several others in which paracentesis was performed by Mr. Pollard at University College Hospital, the symptoms were mainly due, no doubt, to otitis. In several other instances of severe head-retraction the operation has failed to give relief. But in view of the difficulty of diagnosis between otitis and meningitis, and of the fact that the former condition is often a cause of the latter, it is always desirable to have the tympanic membranes incised if the patient be seen at or soon after the onset of the illness, and if the help of an aural surgeon can be obtained. Considering the smallness of the parts in an infant, and the importance of the structures surrounding the tympanum on all sides, this operation should not be undertaken by any one unskilled in aural surgery. [See article "Diseases of the Ear," p. 577.]

When a case of meningitis is seen early it may be desirable to apply a leech behind one or both ears. Occasionally this may obviate the necessity for paracentesis.

With regard to treatment by drugs it is difficult to speak with certainty. We think we have seen decided benefit follow the vigorous administration of mercury; and in some cases thus treated, which were fatal, we were struck by the scantiness of the inflammatory lymph found after death (P.M. 19, 22, and 29), as compared with the amount found in other cases which had not been treated with this drug. But of course it is possible that in the cases treated with mercury the inflammation may have been from the first subacute.

Iodide of potassium in large doses has also seemed to us occasionally of distinct service. If by these drugs we can procure the absorption even of some of the lymph which tends to block the drainage-channel of the brain, we may possibly succeed in preventing hydrocephalus, and so save the child from becoming an imbecile. The importance of this result is so great, and the time within which such results of treatment are possible is so short, that large doses should be given at the earliest possible moment, and frequently repeated. From one to three grains of iodide should be administered every two hours, even to a young infant. These doses will occasionally cause slight nasal catarrh, rarely an acneiform eruption, and still more rarely vomiting; but the condition is so grave that it is necessary to use the largest available dose. Young children bear iodide proportionately much better than adults; and the same is true of mercury. It is almost impossible to produce salivation with mercury in an infant, and difficult in an older child. In both cases the fact is probably due to greater activity of elimination by the kidneys. The mercury may be given in the form of gray powder, a grain three times daily, and also by inunction of half a drachm of mercurial ointment twice daily.

When the inflammatory adhesions have caused permanent obstruction of the drainage-channel of the brain, whether that obstruction be at the issues from the fourth ventricle, in the fourth ventricle itself, or in the iter, it is hardly possible that the mere withdrawal of fluid should prove of any avail. In pronounced chronic hydrocephalus, due to such permanent obstruction, we consider such withdrawal to be useless, and sometimes dangerous. But it occurred to us that if the adhesions were limited to the cerebello-medullary region, it might be possible in the early stage of the disease to break through them and re-establish the drainage-channel. In six cases we requested our colleague Mr. Ballance, who has had large experience of cerebral surgery, to trephine the occipital bone and drain the fourth ventricle. The first of these operations was performed by him in December 1891; the remainder during the year 1892. The same operation was performed subsequently by the same surgeon on two patients under the care of our colleagues the late Dr. Sturges and the late Dr. Hadden. But the operation produces considerable collapse, and in spite of every possible care (including the employment of an incubator to maintain the child's temperature after the operation) the results have been unsatisfactory. And when the obstruction is in the iter, drainage of the fourth ventricle would of course be useless; so also is puncture of the subarachnoid space of the spinal cord in the lumbar region. Drainage of the lateral ventricles we have employed in a few cases, occasionally with some temporary benefit. In one of the six above referred to, trephining of the occipital bone having failed to secure the escape of more than a little fluid, Mr. Ballance inserted a fine trocar through the anterior fontanelle into one of the lateral ventricles. A moderate quantity of cerebro-spinal fluid escaped. Relief of symptoms followed, and the child recovered. Six weeks afterwards it was taken ill at its home with diphtheria, from which it died. The necropsy showed slight cicatricial changes at the posterior base, but no other disease in the brain. It should be stated that this case was one of moderate severity and treated in the early stage with mercury, as well as by operation.

Since 1892 we have felt that the results of operation in the cerebello-medullary region scarcely justified further trial. In cases seen at an early stage of the disease, Mr. Ballance is inclined to recommend the relief of any marked increase of intracranial pressure (usually by puncture of the lateral ventricles), and the subcutaneous injection of a suitable antitoxic serum, if it can be obtained, in the hope of antagonising the microbic activity which causes the meningitis. We add that if such serum-injections are to be of service they must be employed at an early period, before permanent obstruction in any part of the drainage-channel has had time to form. Whether such injections are used or not, it is, we believe, always desirable to continue the free administration of mercury.

Dr. Sutherland and Mr. Watson Cheyne have recently advocated another plan of operation in congenital hydrocephalus. In two cases they have established a permanent drainage from the distended lateral ventricles into the subdural space by making an opening through the cortex, in the hope that the fluid would be absorbed by means of the arachnoidal villi described by Key and Retzius. In both cases the excess of intracranial tension disappeared after the operation. We have thought this plan of treatment worthy of trial in the hydrocephalus caused by posterior-basic meningitis. Mr. Ballance has recently (1898) performed this operation in several of our cases. Most of these were unsatisfactory, but one was a remarkable success.

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OCCLUSION OF CEREBRAL VESSELS

Introductory.—The blocking of cerebral vessels is of importance because of the effects thus secondarily produced upon the nervous structures which they serve. By any vascular occlusion the nutrition of the area of the brain related to the affected vessel is seriously interfered with, and its function impaired or abolished. This effect may be temporary; more usually it is permanent, whether the vessel occluded be a cerebral artery, vein, or sinus. The effect of such an occlusion on other parts of the body depends on the part of the brain affected; and the character of the symptoms in any particular case is determined by the situation and extent of the area from which the blood-supply is cut off, and the nature of the process by which the occlusion is effected. Blocking of vessels, as it occurs in the brain, may be divided into three classes:—I. blocking of an artery by an embolus, usually a piece of fibrin, non-infective or infective, carried in the blood-stream from the heart or some part nearer to it; II. thrombosis in an artery; III. thrombosis in a vein or sinus.

I. Cerebral embolism.—The most common source of an embolus, which finds its way to the brain, is a diseased cardiac valve. The accident does not happen frequently in the first acute inflammation which affects such a valve; it is more commonly the result of a second attack of endocarditis, affecting a part already roughened by old disease. From such a valve some loosely-attached piece of fibrin may easily be separated and carried away in the blood-stream. By far the commonest form of cardiac disease which gives rise to embolism is mitral stenosis; and it will be readily understood how any piece of fibrin detached from the mitral valve may find its way into a cerebral vessel. Aortic valvular disease is a comparatively rare source of embolism, though occasionally it does give rise to it. An embolus may also originate from clotting in the left auricle. The condition likely to give rise to such an occurrence usually depends upon a loss of compensation in mitral disease, leading to a condition in which the auricle is never completely emptied. If, furthermore, a rheumatic condition be superadded, with a consequent state of the blood which renders it prone to coagulate, it will be seen how easily a clot may form, and how a piece of such clot may be washed into the circulation and so occlude a cerebral vessel. The state of the blood during pregnancy and the puerperium, and also during lactation, is such as to render it apt to coagulate; and embolism, the result of clotting in the left auricle, is usually the cause of the hemiplegia which takes place in any of these conditions. Similarly, an aneurysm of any of the large vessels at the base of the heart may be the source of a clot which blocks a cerebral artery. A diseased or roughened vessel wall may likewise give rise to the formation of a clot upon it. Such a clot, or a portion of it, may be small, and narrow the lumen without obstructing it; but it may be swept into the blood-stream, and lead to the complete occlusion of a smaller vessel more distant from the heart. In certain diseased conditions of the pulmonary vein a clot may pass from it into the left auricle, and thence into the circulation; and in morbid conditions of the lung, particles, possibly septic, may pass into the circulation through the pulmonary vein and its branches, and reach the brain, causing not only occlusion of a vessel, but also, if the particle be septic, tuberculous meningitis, or abscess. It is by such a mechanism, no doubt, that old-standing empyema is so apt to give rise to cerebral abscess. And with reference to the other embolic conditions enumerated, it will be understood that if the source of the embolus be a septic one—if, for example, the endocarditis be of the infective variety—the embolus from such a source may, and often does, set up changes at the spot at which it lodges in the brain; changes which depend on its septic character, and are quite independent of the usual mechanical consequences. It has also been said that an inflammatory focus in the pelvis may give rise to embolism; it is unlikely, however, that any particle large enough to cause discoverable mechanical obstruction in a cerebral vessel could traverse the pulmonary capillaries uncaught. Yet in certain cases a patent foramen ovale may permit of cerebral embolism, even if the clot come originally from the venous system. It is also possible that minute organisms might traverse the pulmonary capillaries, which are larger than the cerebral; but in such a case any subsequent cerebral symptoms would be regarded, not as the result of the slight mechanical obstruction, but rather of the infection which is associated with it. In cases in which symptoms of cerebral vascular occlusion occurred in association with pelvic inflammation, these were possibly the effect of an embolus detached from a secondarily inflamed cardiac valve; or of thrombosis resulting in one of the events to be enumerated presently.

The local effects of embolism of a cerebral vessel are, in the first place, a local anæmia in the parts supplied by the vessel. There is also œdema of the surrounding area; and this probably gives rise to impairment of the function of part of the brain not supplied by the blocked vessel: thus the extent and degree of paralysis present in the early stage of embolism is as a rule considerably greater than turns out to be the case after this temporary œdema has subsided. But the blocking of the artery leads also to a degree of venous engorgement and consequent slight rupture of small vessels around the spot, constituting the condition which is known as *red softening*. It is possible, indeed, so far as clinical evidence can inform us, it seems certain, that in some cases, probably those only in which a small vessel is blocked, the collateral circulation is such as altogether to prevent any permanent impairment of the nutrition of the part supplied by the vessel which is blocked; for in a number of cases recovery from the paralysis which follows embolism is both rapid and complete. In other cases, however, recovery is but partial; and in them changes take place in the softened and venously-engorged area which ultimately lead to the formation of a cyst,—the contents of which are fluid and contain blood-crystals, or to the formation of a patch of *yellow softening*. In the description of the clinical consequences of embolism and thrombosis it will be pointed out how frequently, especially in young people with vessels otherwise healthy, recovery takes place in the former to a very considerable degree, or even completely; forming a marked contrast to the usually permanent effects of thrombosis. In the latter case the failure to restore nutrition by collateral circulation is probably to be explained by the nature of the process, and by the degenerated and diseased condition of the vessels by which such collateral circulation would have to be effected.

But from embolism local changes in the vessel which is blocked may also result. If the plug be of a septic or irritating character, its lodgment may lead to inflammatory changes in the vessel wall itself, and consequent extension to other vascular areas or even to other vessels. Again, on the side of the embolus proximal to the heart, more especially if the vessel be atheromatous, its coat may yield, and an aneurysm may be formed; in some cases of embolic hemiplegia ending in sudden death months after the onset of the paralysis, the rupture of such an aneurysm has been recognised as the cause of death.

Embolism is more likely to occur on the left side of the brain; probably on account of the greater facility with which an embolus may enter the carotid of that side. As a matter of statistics, the left side of the brain is affected in about 60 per cent, the right in 40 per cent of the cases. The middle cerebral artery, in its trunk or one of its branches, is the vessel most liable to be affected. The internal carotid itself may be blocked; and the order of affection of the other arteries is—posterior cerebral, anterior cerebral, basilar, and cerebellar arteries. The angle at which the branches from the basilar come off makes the occurrence of embolism in them unlikely. An embolus is most apt to lodge where a vessel bifurcates, and where accordingly a diminution in lumen renders the further passage of clot impossible.

The *symptoms* of embolism are the sudden paralysis; its extent and degree depending on the vessel affected. But, as has already been said, the degree of paralysis in every case of embolism is probably more severe at first than it is subsequently, on account of the temporary impairment of a large area by the conditions consequent on the blocking of the artery. Consciousness may or may not be lost. In the majority of the cases it is lost; and in those in which it is not affected it will probably be found that the vessel blocked is minute, and that the paralysis is slight and probably temporary. It is rarely that the profound coma attending hæmorrhage is present in cases of embolism; nor, unless the embolus be a septic one, are there the febrile indications which accompany the former condition. Convulsion may occur if the vessel blocked be a cortical one. Of course the great indication for differential diagnosis is to be found in the presence or absence of some condition likely to give rise to embolism;

although it must also be borne in mind that the presence of such a condition does not exclude the possibility either of hæmorrhage or thrombosis. The clinical features of the paralysis arising from embolism will be considered in detail along with those resulting from thrombosis.

II. Vascular occlusion from thrombosis in an artery.—The two factors determining this condition are:—(i.) diseased vessels, and (ii.) morbid states of the blood.

(i.) *Diseased vessels.*—The most common disease in vessels—atheroma—is that which is present as a causal condition in most cases of thrombosis. Atheroma is, of course, a senile or degenerative change. It produces roughening in the vessel wall, and on any injury of the endothelium coagulation may take place, and thus start the process of occlusion. This mechanism may become effective only in conditions in which the blood-stream is very slow, or the blood, on account of some alteration in it, has become viscid, or more ready to coagulate than usual. The smaller cerebral arteries are peculiarly liable to be affected with atheroma; and the point at which clotting starts often is at the emergence of a branch from an artery, the lumen of the smaller vessel having become narrowed by atheroma just at this point.

(ii.) Besides the disease in the vessel wall, *the state of the blood* is of importance. Various conditions render it apt to coagulate. In old people it would seem as if great or unusual exertion might have this effect; for one of the commonest conditions under which thrombosis occurs is during the profound sleep which follows unusual fatigue in persons advanced in years. Chlorosis is another condition in which there is great liability to coagulation; but in this condition the clotting not infrequently takes place in the veins or sinuses. During pregnancy, during the puerperium, and apparently after this has passed but while lactation is still going on, clotting may occur; the blood states peculiar to gout and diabetes also favour this change. Although after typhoid fever the veins of the limbs are frequently the seat of coagulation, it may also occur in cerebral vessels—probably in arteries as well as veins. After diphtheria, also, it is not very uncommon to see hemiplegia resulting from vascular obstruction of a cerebral vessel; and this depends, probably, on the blood state, with consequent thrombosis; or it may result, as some believe, from an embolism coming from a diseased valve, the result of associated endocarditis. Further, any condition or disease which leads to very great lowering of the general strength may lead to a state in which thrombosis may readily occur; and it must be remembered that the blocking of cerebral veins may, so far as the clinical condition is concerned, lead to results practically identical with those of arterial occlusion.

The result, then, of vascular occlusion occurring in any such way is to lead to very grave interference with the nutrition of the related part of the brain. It is probable that œdema does not occur in anything like the degree in which it does with embolism, on account of the condition of the blood, as well as of the vessel wall. And in thrombosis there is not the same capacity for collateral circulation, because the diseased vessel is probably in the centre of a district all the vessels of which are similarly affected; so that the recovery from thrombosis, which occurs as the result of atheroma in patients fairly advanced in life, is never so substantial as it may be from embolism occurring in young people. Besides the œdema, which may result, there is also venous turgescence, and the consequent appearance of so-called *red softening.* This red softening usually undergoes certain changes, during which the colour becomes more and more pale until it turns to *yellow softening.* But in some instances a more widespread change takes place, especially if a large cerebral vessel has become blocked; in these large yellow patches —the *plaques jaunes* of the French—are scattered throughout the affected area, and the whole, although softer than usual, is not in the diffluent condition which is the usual result of local thrombosis.

Besides thrombosis arising in connection with atheromatous or other degenerative conditions of blood-vessels, we have also to consider a by no means uncommon result of syphilis, occurring in what is known as the tertiary stage. By such a period no particular time after infection can be indicated, but a stage at which, among other structures, blood-vessels are affected. As one of the phenomena of this stage changes occur in these structures, which result in thickening of the vessel wall, and obliteration, either complete or partial, of the lumen. The change consists essentially in proliferation of the intima, and, associated with this, there is usually some thickening of the adventitia also. The essential features of the process are considered elsewhere ("Disease of Arteries," vol. vi. p. 303); the process, as it concerns us in this place, is narrowing of the lumen, and consequent limitation (it may be entire cutting off) of the blood-supply of a certain area. The effect is interference with the function of the limb or limbs related to the area of the brain affected; and while the usual result is hemiplegia, from a blocking of a vessel on one side of the brain, we may, if such a vessel as the basilar be affected, have interference with the functions of all four limbs, of the facial muscles, and of the parts subserved by the other cranial nerves. In short, the cerebral conditions resulting from occlusion of a vessel or vessels affected with syphilitic endarteritis are not to be distinguished from the results of hæmorrhage, of embolism, or of thrombosis resulting from atheroma; unless it be by a consideration of the mode of onset, the extent of the affection, the age of the patient, the history, and the condition of other viscera.

In many cases of so-called syphilitic hemiplegia the onset of the paralysis is sudden. It is often preceded for some days, or longer, by severe continuous or paroxysmal headache, or by symptoms of general but indefinite illness; not uncommonly with mental symptoms. Convulsion may also precede the paralysis, and this is sometimes unilateral. The paralysis, as has been stated, is usually sudden in its onset, and frequently accompanied by coma, which may last for days, and, indeed, may not be recovered from. There may be evidence of syphilitic processes elsewhere, such as gumma in the liver, nodes in the bones, and such like; and not uncommonly there is marked thickening and hardening of the coats of the radial arteries so that they feel like cords under the finger. The victims of the condition are usually under forty, and it is often impossible to obtain a definite history of syphilis. But given a patient under forty, in whom there is no evidence of cardiac or renal disease, or any other condition likely to give rise to blocking or rupture of vessels, we are justified, even in the absence of any confirmatory history, in assuming, for purposes of treatment at least, that the condition is the result of syphilitic arterial disease. Unless such a case were met with in the early stage, and submitted at once to energetic anti-syphilitic treatment, the hemiplegia is likely to be persistent. When occlusion of an artery sufficiently large to cause hemiplegia has taken place, necrotic changes are as apt to occur in the area supplied by the artery as in any other condition leading to complete blocking. And it may be that much less is to be hoped for from the collateral circulation than in cases of embolism, because it is probable that the other arteries in the vicinity are similarly affected, if not quite so severely. If, however, the patient is at once submitted to energetic treatment, with iodides and mercury, the thickening of the walls of the wholly or partially occluded vessel may be resolved to the extent of permitting the re-establishment of the circulation through it; or perhaps the walls of the neighbouring vessels may be so influenced as to enable them to assume the collateral circulation. If such a happy result be not obtained, then the affected area undergoes necrotic changes similar in character to those following the other kinds of vascular occlusion which have been already described.

Arteries or veins may be blocked as a result of a blood state such as we find present, for example, in rheumatic conditions, in diabetes, in typhoid fever, and in chlorosis. In these conditions the vessel occluded may be an artery or a vein; in the two former usually an artery, in the two latter perhaps more often a vein: the condition will be referred to in dealing with the occlusion of veins and sinuses.

In all forms of vascular occlusion occurring in the brain there is danger of death at the onset; and in many cases this actually occurs, the patient becoming comatose, Cheyne-Stokes respiration perhaps coming on, and death supervening in the course of a few days or even hours. This is particularly true of cases in which the occlusion affects vessels, such as the basilar, which subserve bilateral functions; or those most nearly associated with the vital centres, such as the vertebral: and it may be stated generally that occlusion of a vessel at the base of the brain is always of more serious significance than occlusion of a vessel over the convexity, or of one supplying the subjacent white matter. And in any paralytic condition following supposed vascular occlusion, it is always important to examine into the condition of the cranial nerves, as a definite affection of one of these may furnish important evidence as to the vessel occluded and, through that, to the likelihood of recovery. Such evidence is not infrequently to be met with; for instance, in cases of syphilitic disease of the basilar artery.

The usual result, however, of the occlusion of a cerebral vessel is hemiplegia, uncomplicated with any affection of cranial nerves or nuclei. The initial condition as regards paralysis may be the same, whether the vessel be large or small. In many cases, in two or three weeks, when the effects of the first attack are seen to be passing off, symptoms suggesting an extension of the original mischief, or some secondary effects of it, may take place, and lead to a fatal issue. This is especially true of cases of thrombosis in patients who are the subjects of senile changes in the arteries.

The character of the paralysis resulting from vascular occlusion will naturally depend upon the function of the parts supplied by the occluded vessel or vessels. The ordinary form of paralysis is *hemiplegia*, in which there is weakness of one side of the face and trunk, and of one arm and leg. The whole of one side of the face is usually affected, although the weakness is more obvious in the lower part. The weakness of the affected side of the trunk is not, as a rule, very marked, although it may be distinct enough. The arm, in the great majority of cases, is more paralysed than the leg; and the explanation of these peculiarities is to be found in the hypothesis advanced, in 1866, by Sir William Broadbent, and usually known as Broadbent's hypothesis. The hypothesis is that bilaterally-associated movements are represented on both sides of the brain, and that the closer the bilateral association the more nearly equal is the representation on the two sides of the brain. Thus it is well known that the two lower limbs are much more closely associated in their movements than the two upper. One arm is very frequently used quite independently of the other, while a movement of one leg without some movement of the other is comparatively uncommon; and in the bilateral use of the lower limbs for the purposes of locomotion the association is a very close one. The same is true, even to a greater degree, of the movements of the two sides of the trunk; for it is impossible to move one side of the chest or abdomen without moving the other. Facial movements again are nearly always bilateral; the movements of the two sides of the forehead especially being very closely associated. The orbiculares palpebrarum are less closely associated, although some people find it impossible to close one eye without closing the other. Of the parts about the mouth the association is still close, although less so; much closer, however, than that between the two legs, and still more close than that between the two arms. If, for example, we could suppose a vessel occluded in such a position as to affect the face, arm, leg and trunk areas or fibres equally, the arm on the opposite side would be most affected, the leg less affected, the face still less so, and the trunk scarcely at all—in any obvious way. Similar considerations explain the phenomena of the condition known under the name of pseudo-bulbar paralysis, or double hemiplegia; a condition in some ways resembling bulbar paralysis, but really resulting from cerebral lesions—hæmorrhage or vascular occlusion—affecting both sides of the brain. The condition resembles bulbar paralysis in that swallowing and articulation are affected. The movements subserving these actions are strong in their bilateral association, and consequently, according to Broadbent's hypothesis, in their bilateral representation in the brain; so that a lesion of one side, if it affect them in any noticeable degree, does so but temporarily. If, however, in addition to the weakness caused by a unilateral lesion, we have the weakness produced by a second lesion on the opposite side, it will be understood at once that considerable interference with these actions is not only likely, but almost inevitable; so that with such a double lesion we have a condition similar, in many respects, to that produced by true bulbar paralysis.

Returning now to the usual form of paralysis resulting from vascular rupture or vascular occlusion,—namely, paralysis of one side of the face and trunk, and of the arm and leg on the same side,—such a paralysis may be purely motor. If, however, the area subserved by the posterior cerebral artery be affected, anæsthesia of the same side of the body may be present with affection of the special senses also on this side; the result of a lesion affecting the posterior part of the internal capsule, the "sensory crossway." In such a condition there may be, and indeed there is apt to be, an affection of the fibres which subserve the two halves of the retinæ on the same side as the lesion (therefore the opposite halves of the fields of vision), causing *homonymous hemianopsia*. In such a condition the leg is likely to suffer more than the arm; as the leg fibres in the internal capsule are placed far back, and lie next to the sensory fibres. A similar condition, as regards vision, may also arise from occlusion of a vessel supplying the occipital lobe, and especially the cuneus; and in such a condition, as well as in that resulting from a lesion of the fibres of the optic radiation, there is usually, in addition to the hemianopsia, distinct peripheral restriction of the parts of the field in which vision is preserved; more particularly of that which is on the side opposite to the lesion.

In cases of lesion on the left side of the brain, speech is likely to be interfered with, so as to result in some form of *aphasia* (*q.v.* p. 394); and in left-handed patients a right-sided lesion of the brain may give rise to this defect, while a left-sided lesion in such patients may not have aphasia as one of its results. Dr. Hughlings Jackson has also pointed out that in cases of right hemiplegia in which the patient is not left-handed, and in which there is no aphasia, the leg suffers, as a rule, more than the arm; while the converse is also true that in cases of right hemiplegia, in which the leg suffers more than the arm, aphasia is not so likely to result. The reason for this lies, of course, in the considerable area intervening between the leg area or fibres and the speech area or fibres, as compared with the small space which separates the hand and arm centre and fibres from those subserving speech.

Besides the actual powerlessness which is present in the paralysed limb in hemiplegia, there is usually a considerable degree of *rigidity* also. Three kinds of rigidity are described: (*a*) initial rigidity occurring at the onset of the lesion and ascribed to irritation; (*b*) secondary rigidity which comes on after the lapse of weeks, or it may be of months; and (c) permanent contracture, in which position structural changes have taken place in muscles, and perhaps in joints. This is really a natural consequence of secondary rigidity. The rigidity is a varying condition; that is, it is not always equally well marked. Often in the early morning the limbs are less stiff; and one curious phenomenon is that frequently, while the patient yawns, the hand or arm, at other times rigidly contracted, opens or relaxes, and becomes momentarily limp. The explanation of the rigidity is still obscure. Dr. Hughlings Jackson's view that it is the result of unantagonised cerebellar influx is the most feasible explanation yet offered; although it is difficult on such a hypothesis to understand why it should not be established from the very beginning, when the cerebral influence is cut off [*vide* art. on Spasm, vol. vi. p. 540].

Along with this rigidity there is increased activity of the *deep reflexes.* The knee-jerk is unusually active, not only on the side paralysed, but also on the other. It is really more correct to speak of both sides of the body being weakened, one considerably more so than the other; and thus it is also with the knee-jerks; both are increased, that on the weaker side more than the other. Often a rectus clonus can also be obtained, and usually a foot clonus,—at any rate, on the paralysed side. There is also increased activity in the wrist and elbow reflexes, and often also in the jaw-jerk. The *superficial reflexes* on the paralysed side are usually diminished in activity. At first there is often some difficulty with the bladder—usually a difficulty in passing water, so that retention may result; and, unless this is relieved, overflow incontinence. This, however, as a rule, is a transient phenomenon. The bowels are usually constipated, but there is rarely any incontinence of fæces. There may also be difficulty at first in swallowing, and some interference with articulation; but, except in cases in which there is a lesion on both sides of the brain, these difficulties are usually temporary.

Such are the conditions met with when a vessel is either occluded or ruptured in the cortex, under the cortex, or in the vicinity of the internal capsule. When the lesion affects the crus cerebri, on one side, there is induced not only the hemiplegic condition described, but also an affection of the third nerve on the side opposite to the hemiplegia. The affection may be complete, involving the ocular movements, the levator palpebrarum, and the pupil; and depends upon a simultaneous affection of the third nerve on the one side and the motor tract on the same side, but of course subserving the limbs of the opposite side. If the diseased vessels be those supplying the upper part of the pons, the hemiplegia is of the usual type—face, arm, and leg—all on the side opposite to the lesion. If, however, the affected area be the lower part of the pons—that is, below the point where the tract subserving facial movement crosses to its nucleus—the paralysis is of another type, crossed or alternate hemiplegia; the face being affected on the same side as the lesion, the limbs on the opposite side. With such a lesion the external rectus muscle, on the same side as the lesion, is also liable to be paralysed.

Similarly, there may be wasting and weakness of the tongue on one side, and of the limbs on the opposite side, from a lesion in the vicinity of the hypoglossal nucleus of one side; but such a case of crossed hemiplegia is exceedingly rare.

Certain conditions occasionally present in cases of hemiplegia should be mentioned. Among these is that form of mobile spasm, also named athetosis, which usually occurs in association with the hemiplegia of early life. Fuller reference is made to it in the section dealing with the cerebral palsies of children (p. 741). Although, as a rule, it occurs in such cases, it is occasionally met with in cases in which the onset has been later, especially in such as have been determined, apparently, by injury. In such cases the spasm may be of such extreme violence as to make it necessary to control the affected parts forcibly. In nearly all the cases of athetosis examined after death the apparent determining lesion has been below the cortex; but in some cases in which both sides are attacked with the spasm the time and mode of onset are such as to suggest some injury during birth, such as usually results in submeningeal hæmorrhage. It is also noteworthy that in one case, in which a part of the cortex was removed, the spasm ceased, and remained absent during the time the patient lived. In another case, however, the spasm, after being in abeyance a little time, recurred, although not with such severity as before.

It sometimes happens that much wasting of muscles occurs on the hemiplegic side. Such wasting is, for the most part, the result of inactivity. In cases of hemiplegia occurring in early life the dwarfing of the affected side is not to be regarded as the result of wasting, but of interference with development. In the cases of adult life, however, the wasting is often well marked, more so in left hemiplegia; and is usually associated with joint changes, probably trophic in character. The shoulder-joint is especially prone to suffer, and almost complete immobility of this joint is not uncommon. If no further change have taken place the electrical reactions are found to be unaltered. Sometimes, however, the cords of the brachial plexus are involved in the changes round the joint; in which case we have the usual symptoms characterising nerve inflammation — neuritis — namely, wasting, tenderness of nerve-trunks, loss or impairment of electrical reactions, perhaps the true reaction of degeneration, and glossy skin. Such a condition is often extremely painful, and in many cases must be regarded as of ominous significance.

Another phenomenon, which must also be regarded as of grave import, is the acute sloughing bedsore which occurs on the paralysed side in certain cases of hemiplegia. It is commonly known as *decubitus acutus*, and, as a rule, does not come on until after the paralysis has been established at least a few days. This occurs unexpectedly, and usually is first seen as a reddish spot over the sacrum, the large trochanter, the outer side of the tibia, or the heel. In spite even of the greatest care rapid necrosis may take place; and, when the slough has separated, it is seen that the sore is not merely superficial, but that the deeper parts are affected, it may be down even to the bone. The exact condition underlying this often sudden and unexpected change is not known. It must, however, be one of great gravity, and such severe trophic changes usually initiate a rapid deterioration. Convulsions, accompanied with loss of consciousness, are also of occasional occurrence after hemiplegia. These are doubtless the result of the irritation caused by the initial lesion. Although, as has been stated, convulsion is of occasional occurrence in connection with the onset of embolic hemiplegia, it seems to be of very rare incidence as a later phenomenon in this condition. In some cases in which such convulsion occurs as a post-hemiplegic phenomenon there has been evidence of concomitant kidney disease, so that the attacks may be determined to some extent by a toxic blood state. But it is probable that in the great majority of cases the exciting cause of the convulsions is an unstable condition of the cells in the vicinity of a cortical or subcortical lesion.

In certain cases of hemiplegia mental derangement is a grave symptom. This derangement may be of the nature of acute maniacal excitement, and by some observers such a condition is considered to be strongly suggestive of cortical lesion. In other cases the mental peculiarity may be very slight, and manifest itself only as emotional instability—an undue readiness to laugh or to cry. This condition is more likely to occur in cases in which the lesion is a basal one, or a double one. Between those two extreme conditions varying degrees of mental disturbance may be present. If this be considerable it adds materially to the gravity of the condition; not only because the initial lesion is probably severe, but also because of the additional difficulties in feeding and general management.

Treatment.—The treatment of the conditions of cerebral occlusion which have been referred to in this paper is naturally best considered as two separate problems—A. The treatment at the onset of the condition, which is directed to limit the extent of the morbid state, and to counteract the severe and perhaps threatening symptoms which it may have produced; B. the treatment to be given to the parts of the body whose function may have been impaired by the cerebral lesion; with the object of restoring their function as far as possible, and of preventing the contractures and after-deformities which, as experience shows, are wont to arise.

A. Treatment at the onset.—This must depend upon the diagnosis which has been made. If the case be one in which, relying upon the data already given, the diagnosis of embolism has been made, the treatment which suggests itself is that of almost complete rest; both in order to minimise the danger attending the presence of a block in a cerebral vessel which has already taken place, and to obviate the danger of another embolus becoming detached. If the patient be unconscious, even greater care is needed, in order to prevent any trophic disturbance, or such danger as is apt to arise from a paralytic condition of the sphincters. Retention of urine is very apt to occur, and must be guarded against, if necessary, by the passing of a catheter. The patient will have to be fed regularly with liquid food, chiefly milk and beef-tea, with the addition of alcohol if necessary. When consciousness has been restored, a gradual increase in the quantity and character of the dietary should be ordered, but digestive troubles must be avoided with the greatest care. The bowels will be kept open, of course, but anything like violent purgation must be carefully avoided. If any acute cardiac disorder be present, or if the circulation be deranged in any way whatever, through loss of compensation or otherwise, regard must be had to this in the line of treatment adopted. If the headache be severe, as occasionally it is, a blister, or, still better, a leech, applied behind the ear, or to the temple on the side of the lesion, will often afford great relief.

If the lesion appear to be thrombosis, the treatment, as regards rest, light, easily-digested food, and attention to the state of the bladder, will differ in no essential particular from that recommended for embolism. If there be no reason to suspect syphilis, then the condition is one of clotting occurring in an atheromatous artery; and the three factors which may lead to such a condition have to be borne in mind—the state of the vessel with its roughened wall, the state of the blood with its undue tendency to coagulate, and a weak heart. It is probable that in many cases the indiscriminate use of strong, or at any rate active, purgation in all cases of hemiplegia does infinite harm. It is no doubt desirable to act quickly and smartly on the bowels in order to get rid, if possible, of any toxic influence of the blood which may be present; but it must be borne in mind that, before removing fluid and so perhaps rendering the blood more viscous, a fluid of a bland character should be administered, and in considerable quantity; and along with it stimulants—alcohol and some form of strychnia—are of the highest importance. Often also the combination with them of nitro-glycerine (in one-drop doses of the 1 per cent solution) seems to intensify the action of the strychnia, possibly by giving it more rapid and complete access to the part which it affects. When the immediate danger of any extension of the clotting has been removed by these means, the patient must still rest, have light food frequently administered, and the bowels carefully regulated. Not uncommonly, after a few days, there is evidence of a slight increase in the paralysis, and such a condition is always attended with anxiety. Death has been known to follow such an extension occurring about the third day, and again in more than one case about the tenth day. Convulsions also may occur, but have not necessarily the same serious import, and they are usually well controlled by fair doses (at least half a drachm a day, or more) of a bromide. The tendency to them usually soon passes off, and the bromide may be omitted. If they recur later it is merely as a sequel, and without the more dangerous significance which may be attached to them in the more acute stage of the illness.

If there be any reason to suspect that the symptoms are the consequence of thrombosis occurring in a vessel the seat of endarteritis obliterans, energetic treatment by the administration of iodide of potassium in full doses, and by the inunction of mercury, should be undertaken at once. In some cases which are treated in this way the evidences of cerebral lesion disappear entirely. But it must be confessed that in the *great majority of patients undoubtedly suffering from vascular occlusion from syphilitic endarteritis* the result of the most energetic treatment by antisyphilitic remedies is very disappointing. If the blocking be complete, and the tissue has already lost its blood-supply and is beginning to decay, it is clearly hopeless to try to effect any improvement. It is probably only in cases in which the process of obliteration is not quite complete that improvement may be expected. Nevertheless, every case should have the same thorough treatment, as the condition of other vessels and other organs may be much benefited thereby. The mercury should be rubbed in twice a day, if necessary, until the gums are slightly touched, and this slight affection of the gums should be kept up for at least a month.

B. The second division of the treatment has reference to those measures by which the functions of the parts of the body, which have been weakened by the central lesion, may be restored as far as possible; and the contractures and other deformities which are apt to arise may be prevented or minimised. Stiffness and restriction of movement at the different joints, especially of the upper extremity, is very prone to occur; and these parts should, from the very first, be passively moved at least once a day. If firm adhesion have taken place, as it is especially apt to do at the shoulder, it may be necessary to give an anæsthetic in order to have the joint freely moved. After this has been done, the patient will have to submit to some pain and discomfort in the daily passive movements which will be necessary to prevent refixation. As I have said already, this joint inflammation, or, as some hold, this trophic arthritic change, is sometimes complicated with neuritis, resulting in severe pain, wasting, and glossy skin. The pain in such a condition is most distressing, but great relief may be afforded by the copious application of glycerine and belladonna. When the pain has subsided, the use of the constant current and massage are the means best calculated to improve the nutrition and mobility of the limb. The passive movements recommended will also combat the inevitable tendency to contractures; and a similar purpose is served by gentle faradisation of the extensor muscles of the fingers and wrist, so as to counteract the liability to flexor contractures. In a case in which, either in an early stage or later, any tendency to the occurrence of bedsores shows itself, the use of a water-bed, or water-pillow, is essential; and the most scrupulous care in treatment of the skin must be exercised by the nurse (*vide* vol. i. p. 431). Frequent change of position, the use of "birds' nests" of cotton wool over tender parts, rubbing the back with spirit, and all the varying attention implied in the most assiduous nursing, are of the utmost importance. In many such cases the internal administration of opium seems to exercise a very beneficial effect. As soon as the patient is convalescent, he should be got out of bed daily, and for a longer time each day. He should be encouraged to walk, and to use the affected limbs as much as possible. Improvement, once begun, often goes on to a surprising extent, especially if the patient is young and his vessels healthy. This, of course, is equivalent, or almost so, to saying that such improvement occurs especially in embolic cases. But even in others there is no need to despair, although patient perseverance in treatment is always necessary.

III. Thrombosis in cerebral veins and sinuses.—Besides the occlusion occurring in cerebral vessels already described, in which the vessels affected are for the most part and characteristically arteries, thrombosis may also occur in cerebral veins or sinuses. The blocking which occurs in these structures is either (A) simple or non-infective, the result of some general condition; and (B) inflammatory, or, more properly, infective, due to local infection with septic material.

A. *Simple thrombosis.*—This, the most common form, which is nearly always the result of exhausting or depressing disease, is named marasmic. It occurs in the very young, especially in the first six months of life, or in the very old; or it may occur in middle life, at the end of any long or exhausting illness. The sinuses, probably because of their more rigid walls, their peculiar structure, and the manner in which the veins pour their contents against the stream into some of them—especially into the longitudinal, are more liable to thrombosis than the veins. The longitudinal sinus is that most commonly affected; but, in cases examined after death, the process has usually been found to extend into one or more of the immediately related sinuses as well. The cerebral veins may also be affected; and in some cases, as for instance at the end of an attack of typhoid fever, a vein may be the only vessel blocked. In early life the most common cause is exhausting diarrhœa; and the cardiac dilatation and consequent slowing of the circulation associated with the condition produced are important factors not only in determining the thrombosis but also in causing its spread. Pulmonary affection, such as pneumonia or pleurisy, also seems to have a specially strong tendency to the production of this condition. It occurs, however, not so frequently in the course of the illness as after the subsidence of the acuter symptoms, when the patient is suffering from the consequent exhaustion and prostration.

Morbid anatomy.—As I have already said, the longitudinal sinus is that most frequently affected. Sometimes the affection is solitary; in most cases, however, other sinuses or veins may be involved. In fatal cases the thrombosis, as a rule, extends into more than one vessel. Occasionally a surface vein only seems to be affected, giving rise to the usual symptoms of a focal lesion—unilateral convulsions and local weakness. The clots are dense, resistant, stratified, and non-adherent to the walls of the vein or sinus. They do not, as a rule, completely fill the lumen, and they tend to become organised or absorbed; they rarely disintegrate. By becoming tunnelled, they may permit the re-establishment of the circulation. As a consequence of the thrombosis there is intense congestion of the cerebral veins and capillaries; and numerous minute extravasations from the latter may give rise to a form of red softening similar to that already described as occurring in arterial thrombosis. Considerable œdema may also result. The ventricles may become distended, and a spreading of the fluid into the retro-ocular tissue may give rise to pressure on the eyeballs and consequent exophthalmos. The condition resulting on recovery is not definitely known; probably it is one of atrophy and induration of the affected area.

According to some observers, the form of infantile hemiplegia characterised by paralysis of one side, and preceded by unilateral convulsion and a condition of acute and serious illness, is determined by thrombosis occurring in the surface veins of weakly children. But in some of these cases, at least, there is acute illness with high temperature, and the conditions generally which characterise an inflammatory affection.

Symptoms.—The symptoms are rarely sufficiently definite to indicate a certain diagnosis during life. If an indefinite illness of cerebral character, ushered in by a convulsion or series of convulsions, occur in a patient after prolonged and exhausting diarrhœa or other serious illness attended with great prostration, the probability of sinus thrombosis is great. If this illness be further attended with œdema of the scalp, and if partial convulsions be present, it is to be surmised that the thrombosis in the sinus has extended so as to implicate a cortical vein.

Treatment will be directed to combat the condition which has led to the occurrence of thrombosis. The copious imbibition of stimulating fluid would first be thought of; but if diarrhœa be still present this may be inadvisable. A large hot-water enema may be retained and absorbed; or it may even be necessary to give an intravenous injection of saline fluid. As soon as the alimentary canal can tolerate hot fluids, these should be given in good quantity, and the patient's general strength maintained. If the condition have been in any degree the result of pleural effusion, or other pulmonary condition, this must, of course, be treated in such a way as to relieve the right side of the heart, and so to accelerate the venous circulation.

A sub-variety of simple or non-infective **thrombosis of veins or sinuses** is that which occurs **in chlorotic patients**, usually about the age of twenty. The sufferers are almost invariably women, and the signs of anæmia are, as a rule, unmistakable. The sinuses are the usual seat of the thrombosis, and in this variety, as in the last, the longitudinal is most frequently affected. Small hæmorrhages from engorged veins or capillaries are usually present also. The clots in the thrombosed vessels are usually firm, but may be softened. In these cases there is no evidence of any other disease.

The onset of the *symptoms* is, as a rule, preceded by a period of unusually hard work, and is signalised at the outset by the presence of intense headache, generally accompanied with vomiting. In the more severe cases convulsions come on; these may be unilateral or bilateral, but are usually succeeded by paralysis of one side. When this takes place the condition is usually serious, and ends, as a rule, in coma and death. Occasionally, indeed, recovery from such a condition may take place, but the patient remains more or less hemiplegic. Optic neuritis is frequently present; and it is probable that many of the unexplained cases of optic neuritis occurring in anæmic girls originate in thrombosis of veins or sinuses. In these cases recovery without serious damage to vision is the not invariable rule. In some cases the thrombosis seems to occur in the cavernous sinus, giving rise to proptosis of one eye and limitation of its movements, with optic neuritis. In such a case the clotting may spread and a serious or fatal condition ensue. In some cases, however, recovery takes place, but some interference with ocular movements usually persists, and the sight of the affected eye may be lost or impaired.

B.. *Infective thrombosis.*—This second variety of sinus thrombosis, also spoken of as inflammatory or secondary, to distinguish it from the simple or primary thrombosis already described, occurs much more frequently in adults than in children. The name implies that there is a focus of primary disease to which the sinus affection is secondary. In the great majority of cases the infective focus is in the middle ear; but it may also be found in the nose, in the tonsils, in erysipelatous inflammation of the face or orbit, or in an anthrax pustule on the face. Compound fractures of the skull, in which the wound has become septic, may also be the starting-point of infective thrombosis; and in these conditions the sinus may become involved, either by actual spread of inflammation along the tissue intermediate between the original inflammation and the sinus wall, or by way of infection of a small vein opening into the sinus, setting up thrombosis in it. When the sinus is first affected its inner surface becomes denuded of its epithelium, and the consequent roughness determines the point at which thrombosis starts. The wall of the vessel becomes thickened and infiltrated, so that giving way of the wall, and consequent hæmorrhage, is not a complication to be feared in this condition. But in this way—by direct infiltration around the wall—the area of the process may become definitely enlarged, collections of pus may be formed, and general meningitis may ensue. The existence of a clot in the sinus, especially of a clot the nature of which facilitates softening and breaking down, constitutes even a more serious danger of general infection in the course of the descending blood-current: septic particles are caught in the lung capillaries; and abscess, pneumonia, or gangrene may result. Sometimes thrombi may form lower in the venous stream than the actual place of infection, and the clot may have little or no tendency to break down. In such cases a venous trunk —like the jugular, may be completely blocked, and be palpable, as a hard cord, along the anterior border of the sterno-mastoid muscle.

The *symptoms* of sinus thrombosis are somewhat indefinite, but their broad general features are sufficiently marked to permit the condition to be diagnosed, especially if a source of infection is ascertained. The local symptoms will vary, of course, with the sinus affected; but the most prominent general symptoms are severe headache with vomiting, frequent and severe rigors with profuse sweating, anorexia, diarrhœa, and an irregular temperature, with marked oscillations. Not infrequently also there is œdema of the area of the scalp which is near the thrombosed sinus—over the mastoid, for instance, if the sigmoid or lateral sinus is affected; and there may also be pain, either spontaneous or only to be elicited on pressure. If infarction of the lung have taken place there may be no symptoms during the first twenty-four hours, except some cough and occasional sharp pain in the side. At the end of that time, however, or even earlier, "prune juice" expectoration occurs, and this becomes more brown from the admixture of purulent secretion. Rales are now audible over the chest; the purulent secretion may be abundant, and is very offensive, rendering extensive fumigation necessary. The stools may have the same offensive odour. Pain over the liver may also be complained of, probably from extension of inflammation through the diaphragm. The lungs are found extensively disintegrated, so that one or both are studded with abscesses. The mental condition is usually unimpaired, and may remain so almost to the very end.

In other cases abdominal symptoms—pain, and diarrhœa, pea-soup in character and very offensive, with meteorism—may be present, a condition closely simulating typhoid fever. The symptoms usually come on about the second week, and are accompanied by a dry, foul tongue, and frequently by a dark, measly rash, sufficiently distinct from the rose-coloured typhoid eruption. Headache is also more severe and persistent than in typhoid. Pallor of the face, languor, muttering delirium, and great prostration ensue, and death usually occurs in these cases rather earlier than in those of the preceding type.

In another class of cases, a much smaller one, the symptoms of meningitis — excitement, agitation, twitching of muscles, squint, head-retraction, and delirium—are most prominent. In the great majority of cases, however, the symptoms are made up of a combination of those enumerated in the three varieties, and in the last one especially may be complicated further by those of abscess of the temporo-sphenoidal lobe.

Medical treatment of this condition is of course inefficacious. In some cases surgical measures may relieve, or even cure. For details of surgical procedures with this object the reader is referred to surgical works, especially to the classical work of Macewen, from which much of the preceding description is taken.

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J. T.

ON CERTAIN AFFECTIONS OF THE EAR

OBSERVATIONS ON THE RECOGNITION OF AURAL DISEASE IN MEDICAL PRACTICE

Introductory remarks.

Symptoms: — Deafness, hysterical deafness, tinnitus, vertigo, Menière's disease, pain, mastoid neuralgia, palsy of the seventh nerve.

Causes:—Syphilis, tuberculosis, acute specific fevers.

Intracranial complications.

- I. Abscess of brain.
  - A. Non-localising symptoms.
  - B. Localising symptoms of cerebellar abscess.
  - C. Localising symptoms of abscess of the temporo-sphenoidal lobe.
    - Some general considerations. Rarity of abscess following acute otitis. Importance of vomiting. Slight localising value of optic neuritis. The diagnostic significance of paralysis.
    - Complications of abscess:—(a) Abscess in two situations; (b) abscess with meningitis; (c) abscess with pyæmia; (d) abscess with acute hydrocephalus.
    - Abscess in unusual situations:—(a) In middle lobe of cerebellum; (b) in flocculus; (c) in pons.
  - Diagnosis of brain abscess due to ear disease from other diseases:— (a) Tuberculous meningitis; (b) marantic thrombosis of the sinuses; (c) localised suppurative meningitis; (d) vascular lesions.
  - Some general remarks.
- II. Suppurative meningitis.
- III. Lateral sinus pyæmia.
  - Diagnosis from enteric fever. Remarks on pneumococcus infection.
- IV. Lateral sinus septicæmia.
- V. Lateral sinus sapræmia.

Introductory remarks.—The question is not seldom asked: "Is this an ear case?" A symptom, or group of symptoms, such as vertigo, nystagmus, optic neuritis, vomiting or drowsiness, prompts the above question; and on the truth of the answer the life of the patient may depend. In treating of the diseases of any organ it is desirable, first, to consider the effects produced by morbid processes in the organ itself, the results of which are limited to the production of structural changes which cause impairment or perturbation of function in a greater or less degree; secondly, to keep in view the relation which may arise between diseases of the organ and affections of other parts of the body, or of the body generally, either through the influence which loss or alteration of the function of the organ exercises on the general well-being, or through the effects produced by the extension of the morbid processes themselves to neighbouring or distant parts.

This is notably true in the case of the ear. A due appreciation of all that pertains to its structural integrity and pathological relationships is of the greatest importance on account of the rapidity with which the function of hearing may be damaged, or life itself jeopardised; for the interval during which these disasters can be obviated is of brief duration.

The ancients were cognisant of the danger attending certain forms of ear disease; and even in the present day there is no class of cases which may present more difficulty in diagnosis, or require greater skill in treatment.

The *symptoms* commonly induced by disease of the auditory apparatus —deafness, tinnitus, vertigo, pain and facial palsy—will first be considered, and then the diagnosis of the more important diseases of the ear and complications will claim attention.

Deafness.—Deafness does not necessarily indicate disease of the ear. Tympanic and nervous deafness can readily be distinguished by appropriate tests. Deaf-mutism is either congenital or acquired, usually the former; inheritance is a potent cause; acquired deaf-mutism is generally a sequel of some febrile disease in very early life. Nervous deafness may be due to primary disease or injury of the labyrinth; or to an extension to the labyrinth of inflammation from the middle ear or meninges; or to implication of the trunk or connections of the auditory nerve in meningitis or tumour; or, finally, to disease of the cortex of the opposite temporo-sphenoidal lobe, or of the white substance within it.

Hysterical deafness undoubtedly occurs, and can be diagnosed by the presence of other signs of hysteria; by its sudden onset after emotional disturbance, by the absence of evidence of sufficient cause for the deafness in the auditory apparatus itself, and by the presence, in some cases, of anæsthesia of the pinna and meatus. The hearing may return suddenly. The treatment consists in the treatment of the general disease, and, in certain cases, in the re-education of the sense of hearing. The following illustrative cases may be cited:—

CASE 1.[1]—Female, age 21. Neurotic family. Right membrane incised for otitis following throat inflammation. After all local symptoms had subsided complete bilateral deafness of nervous type rapidly set in. Hearing suddenly returned after four months.

CASE 2.—Female, age 16. Loss of power in arms and legs, deafness in both ears, and aphonia. Deafness dated from the use of Politzer's bag a year before.

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[1] The editor has admitted the records of many cases in this article because they are of kinds which are often very variable and sometimes impossible to diagnose. For the same reason he has made no deletions in respect of articles on cerebral abscess or localisation; to do so would break the continuity of this article, and indeed the over-lapping, if any, is very slight.

The palsy and aphonia were obviously functional. Weir-Mitchell treatment, re-education of muscles of limbs, and Gilles' method of re-education of the sense of hearing brought about complete recovery.

It is not possible to distinguish between disease of the labyrinth and disease of the auditory nerve. If the facial nerve is paralysed, and there is no gross tympanic disease to account for the deafness, it is then probable that the nerves are affected by the same cause along some portion of the path which they have in common. Symmetrical nerve deafness may be due to symmetrical disease of the temporo-sphenoidal lobes, to disease of the corpora quadrigemina or medulla, or to bilateral disease of the internal ear of the nature of chronic inflammation, syphilis, or, possibly, hæmorrhage. Auditory over-action or hyperæsthesia occasionally occurs in hysteria; or, in combination with tinnitus, at the onset of acute diseases and during attacks of headache.

Deaf patients who hear better in a noise are incurable: the exact pathological conditions present in these cases is not certainly known. Deafness, in a greater or less degree, occasionally occurs as an immediate sequel of certain diseases, independently of the occurrence of gross tympanic inflammation; among these rheumatic fever, diphtheria, influenza, and malaria may be mentioned. The cause in some cases may be a neuritis, the result of the disease; but it is not yet possible to distinguish this affection from the possibly permanent effects produced by the toxic action of certain drugs, such as salicylate of soda and quinine; such toxic effects have their counterpart in the loss of vision occasionally induced by the excessive use of tobacco.

Tinnitus.—The symptom is very rare in the young, and generally appears after middle life; it is not infrequent, however, amongst students, and in them is often associated with excessive smoking. In these cases slight deafness is often present also,—from Eustachian obstruction, due to irritation of the naso-pharynx or to the presence of small adenoids. Tinnitus is common in old persons, and is often combined with some amount of deafness from labyrinthine disease or other senile changes; gouty patients, or those prone to migraine or neuralgia, often suffer from it. Subjective sounds are of various kinds, and may depend on various causes:—on accumulation of wax, on the presence of foreign bodies in the meatus, on disease of the tympanum, on exposure to a loud sound, on falls on the head, on vascular changes, on anæmia, and on many central nervous conditions. Intermittent tinnitus is of better prognosis than continuous tinnitus. Paroxysmal vertigo may be heralded by paroxysmal tinnitus, and then the noise becomes distressingly intense. As a rule deafness is present with tinnitus, the cause of the former being also the cause of the latter. Conditions of the middle or outer ear act only by the influence they exert on the labyrinth. Elaborate sounds without any discoverable deafness may be of central origin.

Vertigo.—The sense of equilibrium is the appreciation of the position of the body and its relation to other objects in space; when this sense is disturbed vertigo results. In order to maintain equilibrium something more is required than the senses of sight and touch, and the muscular sense. In the co-ordination of voluntary movements the semicircular canals have important functions. Excitations of the nervous endings in the ampullæ give rise to afferent impulses which determine the issue of efferent impulses to the muscles. Disease or injury of the semicircular canals, implication of the vestibular portion of the auditory trunk in disease, by pressure or otherwise, or of its fibres of origin in the cerebellum, will cause the loss of normal ampullar impulses, and throw the co-ordinating function out of gear.

Vertigo is sometimes associated with visual sensations, nervous exhaustion, gastric disturbances, disease of the cerebellum, or tumour beneath the tentorium pressing on the auditory nerve. It is important not to assume at once that the vertigo in a given case is of aural origin. If there be no loss of hearing the labyrinth is probably affected only indirectly; and even if deafness and tinnitus are present, the disease may still not be in the internal ear. In a great variety of diseases of the ear the vertigo may be slight and transient, or it may be constant, and associated with vomiting and tinnitus. It occurs in consequence of pressure on the tympanic membrane, as for instance by cerumen, or syringing. It reaches its acme in diseases and injuries of the membranous labyrinth. Illustrative case :—

A nurse was syringing the ear of another nurse with a brass syringe furnished with a long and fine nozzle. The instrument slipped, perforated the upper posterior quadrant of the drum, and impinged with force on the inner wall of the tympanum. The girl fell at once, and for thirty-six hours suffered from extreme vertigo, frequent vomiting, and loud tinnitus. Deafness was absolute. Six months elapsed before recovery was complete.

Caries of the inner wall of the tympanum, even if limited and superficial, may induce vertigo, especially if it involve the inner boundary of the channel leading between the antrum and the attic; for then the horizontal semicircular canal is most likely to become implicated. In this sense vertigo complicating chronic otorrhœa or cholesteatoma of the antrum or attic is of serious import, and we cannot expect the treatment of the disease by operation to be followed immediately by the cessation of the giddiness. Extreme vertigo, a rolling gait, and lateral nystagmus may all be due to acute otitis media: the hyperæmia around the intense inflammation in the tympanum probably affects the vestibular termination of the auditory nerve, and possibly also the meninges in the posterior and middle fossæ.

Illustrative case :—

Male, age 29. Was seen a fortnight after an attack of influenza. During this fortnight he had suffered from pain in the right ear, extreme vertigo, frequent sickness, and severe frontal and occipital headache; on examination the right meatus was greatly swollen, and there was a red blush behind the pinna, and also in front of the meatus. There was marked nystagmus, especially on looking towards the left, the eyes were suffused, the optic discs congested, the pain in the ear and head severe, and the giddiness induced a rolling gait, which made it impossible for him to cross the room without assistance. The left knee-jerk was absent. One hour after free incision of the meatus and membrana tympani a drachm of pus escaped into the mouth, and all the symptoms disappeared; up to the time of the operation there had been no external otorrhœa. The escape of pus by way of the Eustachian tube is probably explained by the obstruction of the meatus.

Menière's disease.—This name is generally given to that form of aural vertigo which is intense and paroxysmal. The patient may be unable to rise or move during the attack, and he suffers from frequent vomiting and tinnitus. In vertigo the higher cortical centres are probably affected by the morbid process in the internal ear, inducing afferent impulses of irritation allied to those which must travel upwards in the case of tinnitus; and, as in tinnitus so in vertigo, the perception of the morbid state is a function of the higher cortical centres.

Probably in a few cases the occurrence of sudden labyrinthine hæmorrhage is the exciting cause. In most cases one ear is affected, and on examination partial nervous deafness can be made out. The attacks come on as suddenly as the gastric crisis or lightning pains of tabes, and the typical symptoms may arise from central lesion or tumour pressing on the auditory nerve or direct disease of the labyrinth.

In a patient who was anxious to submit to any treatment which offered the prospect of immediate cure I proposed to divide the auditory nerve, having found by experiment that the difficulties of the operation were by no means insuperable; but as no attack took place during the eight weeks the patient was in hospital the operation was not done. In this relation it is important to note that division of the auditory nerve in the monkey results in falling towards the same side as the lesion, but in tumour of the auditory nerve in man (slow division) no such symptom has been described.

In the case of a gentleman suffering from chronic deafness of the right ear, and who was suddenly seized with persistent vomiting, extreme vertigo, distressing tinnitus, and total deafness in the right ear, there was a history of syphilis; the administration of specific remedies restored him to health. Three previous attacks had occurred, and, as the deafness and other symptoms disappeared, the cause was thought to have been one of gumma involving the posterior surface of the petrous bone, which, by pressure on the auditory trunk, had induced the symptoms; and that the delicate structures of the labyrinth had not been directly involved.

The prognosis of Menière's disease is so far good that, under tonic treatment, the attacks become less frequent; but four or five years may elapse before they finally pass away. In the meantime the condition of the patient in the attacks is most distressing, and he is always in fear and danger of them. Dr. Bristowe said that when the deafness becomes absolute,—when, in other words, the function of the auditory nerve or centre is abolished,—then the manifestations of Menière's disease, which depend on the conducting power of the vestibular portion of the nerve, also cease.

Pain.—Independently of the more common causes of pain in the ear—such as the pressure of hard wax and foreign bodies on the drum, obstruction of the Eustachian tube, and inflammation of the drum—it is important to recognise that severe pain in the ear is more often indicative of purely local mischief than of extension to the meninges or brain; and that the absence of pain in the ear is no proof that a local suppurative inflammation is not extending and giving rise to some serious general illness. Neuralgia of the mastoid region, commonly called pain in the ear, may be met with years after the obsolescence of all inflammation in the temporal bone, and is strictly comparable to the pain experienced in other bones from the occurrence of sclerosis after chronic inflammation or abscess; and it has to be treated in the same way, namely, by incision of the bone.

Facial paralysis.—It is unnecessary in this place to indicate the difference between cortical and peripheral facial palsy (vol. vi. p. 798). It is well known that when suppurative disease in the temporal bone has lasted for some time, especially in children, the facial nerve may become involved in the inflammation and be paralysed. This facial palsy may precede as well as accompany pain and external discharge; and, especially in very young children, it is by no means rare for facial paralysis to result from inflammation of the tympanum without any sign of inflammation of the drum. The thinness or incompleteness of the aqueduct offers an explanation of the facial palsy which not infrequently accompanies otitis media in infants, the liability to which certainly decreases as age advances. In 658 cases of suppurative otitis it occurred four times without external otorrhœa. The diagnosis in these cases, when there is no reddening of the drum, can only be made by free incision of it.

It is very common at autopsies of infants to find pus or muco-pus in the tympanum without any symptom, referable thereto, having been observed during life. I have often seen cases of posterior basal meningitis with retraction of the head in which the drum showed no sign of inflammation, but in which, on incision, pus escaped. Many cases of acute convulsions with fever in young infants are due to the presence of pus or muco-pus in the tympanum, and when the membrane is incised or ruptures convalescence rapidly ensues.

Illustrative cases:—

Case 1.—Age 1½ years. Pain in the left ear one week, then left facial paralysis. Pain continued for another seven days, when the membrane ruptured and pus was discharged.

Case 2.—Male, age 17 years. Pain right ear, four days. Fifth day: complete facial paralysis. Sixth day: drum red and bulging, no external otorrhœa, but pus seen oozing from pharyngeal end of Eustachian tube. Treatment: free incision, leeches, fomentations. Result: profuse otorrhœa, which rapidly subsided; recovery from facial palsy in three months, under electrical treatment.

CASE 3.—(Not under care of writer.) Male, age 40. Influenza, double acute otitis media, double facial palsy. On the right side, free incision of membrane, antrum opened, and antral and attic cavities washed out with strong antiseptic solution. On the left side, free incision of drum only. Result: on the right side, complete recovery from both deafness and facial palsy; on the left side, tympanum destroyed, deafness and facial palsy permanent.

Syphilis.—*Hereditary syphilis* usually attacks the ear at puberty, and the ear affection is often present with, or shortly after, an attack of specific interstitial keratitis; the keratitis yields to treatment, the deafness does not. In hospital practice I have seen the tympanic membranes becoming white and thick while the cornea was clearing. This interstitial myringitis is comparable with the disease of the cornea, and may spread inwards from the circumference of the drum. The whole tympanum, and the labyrinth also, may be affected in the same way; and it is common to find nervous deafness coming on while the tympanic disease is in progress.

Acquired syphilis.—Condylomata may appear in the meatus, and the contraction of ulcerative processes in the pharynx may close the mouth of the Eustachian tube, when the ordinary secondary changes will take place in the drum and other parts of the tympanum. The labyrinth may be involved in the late secondary or tertiary stages, the onset being gradual or sudden; in some cases antisyphilitic remedies may effect a cure.

Tuberculosis.—Tuberculosis of the tympanum often occurs in the course of chronic otorrhœa in early childhood. It has a tendency to spread to the mastoid, and to cause mastoid abscess; and, as in other tuberculous affections of bones in the child, large sequestra are often removed during operation. Tuberculosis of the tympanum may cause infection of other parts, as of the glands of the neck, of the membranes of the brain, or of the lungs; or a general miliary infection may arise, which may be called lateral sinus tuberculosis, as, in this event, the bacilli probably travel in the blood-stream to distant parts. In adults tuberculous ulceration of the tympanum is usually associated with disease of the lungs or larynx. It is more painful than non-tuberculous otorrhœa, and solution of lactic acid is useful in treatment.

Acute specific fevers.—Scarlet fever, measles, diphtheria, and influenza are frequently the exciting cause of acute otitis. The patients who suffer in this way from acute otitis are usually those with adenoids. The importance of the early recognition, and prompt and efficient treatment of the inflammatory conditions of the middle ear that occur in the course of these acute specific fevers, or shortly after them, cannot be too strongly insisted upon; as great and irreparable damage to the delicate structures of the tympanum and the consequent impairment of hearing are a frequent consequence. If the affection is bilateral the serious after-effects are, of course, much intensified. Partial deafness on both sides is one of the greatest hindrances to good education.

A free incision, not a mere puncture, should be made in the posterior half of the membrane, so as to give free exit to the pus; if after a few days the free discharge does not diminish by the use of the ordinary antiseptic measures, the delicate structures in the tympanum are jeopardised. An opening should then be made into the antrum, and, avoiding all mechanical interference with the tympanum, that cavity should be flushed with an efficient antiseptic solution. The result is to arrest the secretion of pus, and to save the tympanum from further damage. Influenza often causes very acute otitis in the adult, and requires the treatment described above; the pus of this disease may infect the mastoid, not by way of the antrum, but by an infection which spreads through the posterior wall of the osseous meatus into the middle portion of the mastoid. Such a condition obviously calls for operation, and the prognosis is favourable for rapid healing of the wound, and speedy recovery of hearing.

Illustrative cases :—

Case 1.—Female, age 13 years. Double acute otitis. Influenza extremely prevalent at the time. When seen, drowsy, in much pain, drums bulging and red, temperature 104°. Free incision made in both drums; profuse discharge. Temperature remained high, and discharge continued profuse for some days; antrum on each side opened, and tympano-antral cavities flushed with a strong antiseptic; the fluid escaped through the incisions in the drums and down the Eustachian tubes. Convalescence rapid, hearing returned in so perfect a degree that patient now, many years after the operation, is an admirable musician.

Case 2.—Female, age 20. Influenza, left acute otitis, incision of membrane, leeches and fomentations; discharge continued with pain and fever for two weeks. When seen, swelling along posterior wall of osseous meatus, very slight œdema below posterior border of mastoid. Operation; carious track found, from opening in posterior wall of osseous meatus to digastric groove, abscess beginning to form in neck beneath deep cervical fascia. Rapid and complete recovery, practically no loss of hearing.

Case 3.—*Mastoid disease following influenza, no external signs.* — Male, age 1½ years. Influenza; profuse right otorrhœa two weeks, neither swelling, œdema, nor evident tenderness over mastoid. Operation: diffuse suppuration of cellular mastoid. Complete recovery.

Case 4.—Male, age 3½ years. Influenza; enlarged tonsils, and adenoids; very acute double otitis. Two days later, temperature 104°, pulse rapid, child drowsy, sick several times, drums red and bulging. On incising right membrane, which was under such pressure that it spurted on to the head mirror, at least half a drachm of pus escaped. On incision of left drum pus filled meatus. Rapid recovery from the acute condition, but slight chronic otorrhœa not arrested until adenoids were removed.

Case 5.—Male, age 15 years. Measles; during convalescence sudden onset of severe pain in both ears, with fever. Two days later, temperature 103°, profuse right otorrhœa, left ear severe pain, but no discharge, slight œdema over mastoid. Next day, as condition was unchanged, medical attendant opened left mastoid and scraped out the tympanum with the membrane and ossicles.

A few hours later when I saw the case the proposal was made to plug the sinus and ligate the vein. This was negatived. Result of treatment: complete tympanic deafness on left side. This case illustrates the evil resulting from the performance of too extensive an operation in acute otitis. The proper treatment was free incision of membrane and, if necessary, the drainage and flushing of the tympano-antral cavities by a surgical opening into the antrum.

Swelling over the mastoid.—A swelling over the mastoid in acute or chronic otitis media must not be attributed immediately to inflammation of the bone beneath; it may depend on suppuration of a mastoid gland, or on the escape of pus from the meatus over the mastoid between the cartilaginous and bony canals, as well as on the passage of pus through the mastoid on to the surface.

INTRACRANIAL COMPLICATIONS OF EAR DISEASE.—Suppurative disease of the middle ear is a frequent cause of intracranial inflammation, the most important varieties of which are: I. Abscess of brain, II. Suppurative meningitis, and III. Lateral sinus pyæmia.

I. Abscess of brain.—A. *Non-localising symptoms.*—Over 40 per cent of all cases of abscess of brain are secondary to disease of the middle ear. The site of abscess, with few exceptions, is either in the lateral lobe of the cerebellum, or in the temporo-sphenoidal lobe, in the immediate proximity of the bone disease (posterior or anterior surface of the petrous) from which the infection has arisen. Cerebellar abscess is twice as frequent as abscess in the temporo-sphenoidal lobe. It appears, too, that abscess in the right cerebellar hemisphere is twice as frequent as is abscess in the left; and that abscess in the right temporo-sphenoidal lobe is more common than in the left. Abscess of the brain, secondary to ear disease, is most common between the ages of ten and twenty. The symptoms common to all cases of brain abscess are headache, vertigo, photophobia, purposeless vomiting, slow cerebration, drowsiness, optic neuritis, low temperature, slow pulse with irregular rhythm, slow respiration, foul breath, constipation, emaciation, pallor of face, and expressionless countenance. These cases usually follow long-continued otorrhœa, and have, commonly, a history of sudden onset of illness, with earache, a little fever, and sometimes shivering.

A few exceptions to the rule that intracranial complications follow chronic otorrhœa only may be noted; for example, some cases of an acute otitis media in the young, arising in the course of an acute specific fever which is shortly followed by cerebellar abscess.

Illustrative cases:—

CASE 1.—Male, age 7 years. April 7th, scarlet fever began; April 24th, first sign of right otitis media; May 18th, mastoid symptoms; May 24th, mastoid operation; August 16th, death from cerebellar abscess.

CASE 2.—Female, age 16 years. Enteric fever September 10th, very severe attack. Double otorrhœa October 6th, preceded by deafness dating from the beginning of the illness. December 1st, right membrane healed, with large perforation; left membrane similar perforation, through which pus was discharging. Patient apathetic, very feeble, temp. 101°, pulse 120, variable; had vomited occasionally. The question that had to be determined was this: was the patient, who was convalescing slowly from a severe attack of typhoid, in the state known as post-febrile fatuity, in which vomiting might occur, or was she suffering from an intracranial complication of otitis media? During the next few days vomiting became more frequent, but there was neither paralysis, anæsthesia, nor optic neuritis. On December 12th, some swelling along upper part of left jugular. Operation: purulent thrombus in left lateral sinus, condition unrelieved. Death, December 15th. *Autopsy.*—Left cerebellar abscess.

Note.—The vomiting and mental condition were due to the abscess, the influence of which was also seen in the temperature and pulse records. These were not characteristic of either lateral sinus thrombosis or brain abscess. The temperature varied from 100° to 102°, and the pulse from 100 to 130. Both would have been higher in uncomplicated pyæmia and lower in uncomplicated abscess. It is assumed in the above cases, and is certainly true of the typhoid case, that prior to the acute otitis no inflammation of the ear had occurred. The histories are definite on this point, but in hospital practice the neglect of chronic otorrhœa is so common, and the memory of past events so imperfect, that such statements must not be readily accepted.

In certain uncomplicated cases symptoms are present which are pathognomonic of the site of the lesion:—

B. *Localising symptoms of abscess of the cerebellar hemisphere.*—(*a*) Abnormal motor phenomena:—(i.) Forced position in bed. The patient tends to lie curled up in bed, with the limbs flexed and the side of the face corresponding to the lesion uppermost. (ii.) Conjugate deviation of the eyes to the side opposite to the lesion. (iii.) Horizontal nystagmus, the jerks being most obvious when the patient looks away from the side of the lesion, the jerks being towards the side of the lesion. (iv.) Marked paresis of the upper limb on the same side as the cerebellar lesion. (v.) Weakness of both lower limbs. (vi.) In rare instances muscular rigidity of the limbs, or spasm of the face and limbs, on the side of the lesion. (vii.) Exaggerated knee-jerk on the side of the lesion. (viii.) Rotation; cerebellar gait; a tendency in walking to face toward the side of the lesion, and to fall towards the side opposite to the lesion. (*b*) Abnormal sensory phenomena:—(i.) Deafness, if present, is on the same side as the lesion, and is due to local causes. (ii.) No cutaneous anæsthesia of the face, trunk, or limbs. These symptoms closely resemble the effects produced by the removal of one lateral lobe of the cerebellum in the monkey.

C. *Localising symptoms of abscess of the temporo-sphenoidal lobe.*—(*a*) Abnormal motor phenomena:—(i.) Paralysis of the third nerve, in whole or in part, on the same side as the abscess; a stabile pupil on the same side as the abscess is an important sign. (ii.) Paralysis on the side of the body opposite to the lesion, which may be of cortical origin, the face being first affected, and then the arm and leg; or of internal capsule origin, the sequence of paralysis being reversed, and the parts being involved in the order—leg, arm, face. (iii.) Occasionally convulsions of the face and limbs, beginning on the side opposite to the lesion. (iv.) Exaggeration of the deep reflexes on the side opposite to the lesion. (*b*) Abnormal sensory phenomena :—(i.) Deafness on the side opposite to the lesion, due to implication of the posterior part of the temporo-sphenoidal convolution. (ii.) Anæsthesia of the face, trunk, and limbs on the side opposite to the lesion; either of internal capsule origin, when complete hemianæsthesia is present, or of cortical origin, when there is partial hemianæsthesia with deficient power of localisation and loss of muscular sense—this being most marked in the limb corresponding to the cortical area chiefly affected. (iii.) Aphasia; if the abscess is on the left side in a right-handed patient; this may be of motor or sensory variety. (iv.) There is also a peculiar mental phenomenon recognised as pathognomonic of lesion of the temporo-sphenoidal lobe, namely, "the dreamy state," which may occur as the result of either a right or a left temporo-sphenoidal abscess.

The records of cases of abscess of the brain, secondary to ear disease, show that, in many, an incorrect diagnosis was made during the life of the patient, or none at all; and in more than one instance an operation has been done in the wrong place, or even attempted on the wrong side. The lack of definite localising symptoms is most striking, especially in cases of cerebellar abscess; and if any conclusion can be drawn from personal experience and published cases, it is that life is often lost from the condition of the brain escaping recognition or localisation. Perhaps the most important sign of abscess is *vomiting*. Without this symptom no brain abscess could be acutely extending. In cerebellar abscess we expect marked vertigo, frequent vomiting, occipital headache, and sometimes retraction of the head, which depends on the coexistence of meningitis of the posterior fossa. Cerebellar incoördination is a prominent symptom when the abscess is pressing on the middle lobe or extending into it. The difficulty of diagnosis is often increased by the patient not being accurately examined till the intelligence is abolished, or so impaired as to make a complete examination impossible. The history of ear trouble is often unknown to the friends of the patient, especially in hospital practice; and their story, too, of the days of illness previous to admission is for the most part clinically valueless.

Optic neuritis may or may not be present in a case of brain abscess, and it is not often of importance in localising the site of the abscess. It is possibly more common in cerebellar than in temporo-sphenoidal abscess. It may be more intense on the side of the abscess than on the other, and it may be present only on the same side as the abscess. It has been shown that optic neuritis, of intracranial origin, is due to an extension of inflammation to the sheaths of the optic nerves from inflamed pia mater; and that in these cases, therefore, optic neuritis is pathognomonic of basal meningitis. Basal meningitis may be present which is not visible to the naked eye after death, yet which had nevertheless produced optic neuritis. Thus it is that some cases of chronic otorrhœa are complicated by optic neuritis. The question then arises whether there be any other complication—latent brain abscess, for example—besides the basal meningitis? I have observed several such cases. The optic neuritis is slight, and may last for several months without impairing sight. It subsides when the local disease, from which it arises, is efficiently removed. There is occasional headache. In one case occasional vomiting occurred; but in this the patient, who for a time refused operation, had continuous slight headache, was feeble, and sallow of countenance; and the symptoms gave rise to the suspicion that a latent brain abscess was present. Optic neuritis may, of course, occur also in cases of chronic otorrhœa complicated by mastoid suppuration, or subdural abscess; and is an indication for immediate operation. Optic neuritis, with a stabile pupil in the same eye, has sufficed to localise a temporo-sphenoidal abscess. The stabile pupil is the main factor in the diagnosis. Optic neuritis confined to the side of the abscess is more likely to be present in temporo-sphenoidal than in cerebellar abscess.

The forced position assumed in bed does not enable a diagnosis to be made between cerebellar and temporo-sphenoidal abscess. The typical position in cerebellar abscess has been mentioned. In the last two cases of temporo-sphenoidal abscess observed by myself the patients either assumed the supine position, or lay on the side with the side of the face corresponding to side of lesion against the pillow. Frequent depression of lower jaw, and yawning, gaping, or champing movements of the jaw, have been observed in lesions below the tentorium, including cerebellar abscess.

The site of headache is not often of value in locating a brain abscess. Fixed pain in the region of the abscess has, however, been observed, and also tenderness on palpation and percussion.

Examination with speculum and probe may determine the direction the disease is taking, either into the middle or posterior fossa. The same point is often apparent during the mastoid operation, and, when doubt exists, may help in the location of a brain abscess. The quantity of pus escaping may be so great as to afford conclusive evidence that it must be coming from a large cavity, which can only be intracranial. Both cerebellar and cerebral abscesses have been known to drain in this way.

In my last case of temporo-sphenoidal abscess there were, among other signs, sensory aphasia and a large continuous flow of pus from the left auditory meatus. The pus was found to be coming from an abscess in the temporo-sphenoidal lobe, through a carious opening in the roof of the attic. The main difficulty in the diagnosis of cerebellar abscess is due to the absence, in the majority of cases, of the characteristic paralysis. It would seem from the developmental, clinical, and experimental points of view that the lateral hemisphere of the cerebellum has to do with the innervation of the limbs. Whatever be the direct or indirect nature of this connection, the efferent impulses must come from the cells of the cortical gray matter and of the dentate nucleus. Commonly a cerebellar abscess is in the anterior and outer part of the hemisphere, when the dentate nucleus will escape; and but few of the fibres passing from the gray matter to the dentate nucleus will be involved. When the abscess is large, or when it is placed near the middle lobe, more of the fibres coming from the cortical gray matter are cut off, or the dentate nucleus itself is destroyed; and then the characteristic paralysis is present.

The conjugate movement of the eyes to the opposite side is produced by the weakness of the muscles which may be considered as belonging, functionally, to the same side as the abscess; the ocular paresis is, therefore, comparable to that which obtains in the muscles of the limbs on the same side as the lesion.

Nystagmus occurs in disease of so many other parts of the brain which involve weakness of the ocular muscles, that in the diagnosis of cerebellar abscess this sign is only of value when associated with other characteristic signs. Horizontal nystagmus, nevertheless, is often associated with disease of the lateral lobe of the cerebellum, while rotatory nystagmus points to invasion of the middle lobe. The knee-jerk, on the same side as the cerebellar lesion, is exaggerated; this is exactly what we should expect in association with the weakness of the limbs on that side. The cerebral tonus being low, the spinal tonus is increased, restraint of the local action of the cord being removed.

It has been suggested that if a tuning-fork cannot be heard on the diseased side, the caries, having destroyed the internal ear, has probably reached the posterior surface of the petrous bone; and that under such circumstances it is likely that the abscess is in the cerebellum rather than in the temporo-sphenoidal lobe. That this is not an unfailing test of the site of abscess is shown by the fact that in the last two cases of temporo-sphenoidal abscess under my own care the tuning-fork could not be heard, pus having destroyed the internal ear.

In cerebellar abscess the lateral ventricles often become distended, and the percussion note over the pterion is markedly altered, the resonance being greatly increased. This has been described as a diagnostic sign of cerebellar abscess, under the name of *the differential cranial percussion note.*

In abscess of the temporo-sphenoidal lobe *paralysis* is almost certain to occur, sooner or later, on the side opposite to the lesion. Convulsions of the opposite side of the body sometimes replace or precede the paralysis. The importance of the partial or complete paralysis of the third nerve on the same side as the abscess has already been pointed out. Anæsthesia of the opposite side of the body, and possibly also hemianopsia from invasion of the optic radiation,—neither of which symptoms are present in cerebellar abscess,—may also be present. Since the cortex at the front end of the hippocampal gyrus is especially connected with the sense of smell, it is not surprising that in a case of temporo-sphenoidal abscess the patient complained, many days before the definite symptoms appeared, of a disagreeable odour, which he referred first to one object and then to another.

Abscess in the cerebellum and in the temporo-sphenoidal lobe, like abscess elsewhere in the brain, may be latent, producing only general symptoms of ill-health, until excited to renewed activity by a febrile attack, by a blow on the head, or by some minor operation such as the removal of a polypus.

A voracious appetite is frequently noticed in convalescence from brain abscess, and is one of the best indications to the medical attendant that the patient is making satisfactory progress.

About 80 per cent of abscesses secondary to ear disease prove fatal before the end of the fourth week; 40 per cent in two weeks, and less than 10 per cent in one week from the onset of acute symptoms.

Cases illustrating latency of abscess:—

Case 1.—(Not under care of writer.) Female, age 23. Left otorrhœa many years. January 27th: polypus removed, five days later vomiting, vertigo, and occipital headache, temperature varying from subnormal to 101°; condition remained much the same with intermissions till March 23rd, when mastoid operation was done. Left hospital April 22nd, general condition not much improved. Readmitted July 20th, had been fairly well with occasional recurrence of the symptoms. During last fortnight had been worse: headache, sickness, and marked loss of flesh. On readmission much emaciated, headache and vomiting, temperature 98°, pulse 60, left arm (?) weaker than right, left knee-jerk more brisk than right. July 21st: left cerebellar abscess evacuated, died September 14th. In the interval temperature above normal, pulse rapid, delirium, occipital pain and vomiting. *Autopsy.*—The left cerebellar hemisphere was nothing but a shell of softened gray matter. This case illustrates the danger occasionally attending the removal of an aural polypus, and also affords an example of the long-continued and subacute course of a cerebellar abscess, lasting eight months from the first symptoms to the termination.

Case 2.—*Latency of abscess and renewed activity after the removal of a polypus.*—Female, age 21. Chronic left otorrhœa many years. Polypus removed. Vertigo, staggering gait, and headache for a few days. Three months later another polypus removed; next day headache, vomiting, vertigo, torpor and shivering, temperature 98°, pulse 80. Lies on right side. Twenty-four hours later mastoid operation and evacuation of cerebellar abscess. Death. *Autopsy.*—Acute abscess had been opened in inner and anterior part of left cerebellar hemisphere. No meningitis. No thrombosis of sinuses.

Complications of brain abscess.—The preceding account refers to uncomplicated cases of brain abscess, but in complicated cases the problem of diagnosis is far more difficult.

(*a*) *Abscess in cerebellum and in temporo-sphenoidal lobe.*—The simultaneous formation of abscess in the two situations must confuse the symptoms, and probably render the diagnosis impossible. Unless one abscess form after the other, and the case be most carefully observed from day to day, successful treatment would be well-nigh hopeless. It has happened in practice that both abscesses have been opened.

(*b*) *Abscess with meningitis.*—The symptoms of abscess will be modified or controlled by those of meningitis, according as the abscess or the meningitis is the more prominent disease. In abscess complicated with meningitis the temperature is relatively high; the pulse is quick; delirium, convulsions, and optic neuritis occur early; pain in the head is severe, and retraction of the head may be present, together with vomiting, squint, and irregular respiration.

(c) *Abscess complicated by sinus pyæmia.*—The lateral sinus is often involved in cases of cerebellar abscess, the abscess in the cerebellum being secondary to sloughing of the wall of the sinus. The symptoms therefore are, first, those of pyæmia, and, secondly, those of abscess. As the abscess increases, the mental state becomes impaired, and the lower temperature and slower pulse of abscess replace the oscillating temperature and rapid pulse of pyæmia.

(d) *Abscess complicated by acute hydrocephalus.*—Acute hydrocephalus is no uncommon complication of cerebellar abscess. If an abscess burst or leak into one of the ventricles, general purulent infection of the ependyma occurs.

Illustrative Case.—F., age 11 years. Discharge from left ear three years. When seen all the type symptoms of cerebellar abscess were present, with the exception that, instead of conjugate deviation, skew deviation of the eyes was observed, such as results from the removal of a cerebellar hemisphere in the monkey. Operation forthwith; respiration ceased as the patient was put on the table; large abscess of the cerebellar hemisphere evacuated during the performance of artificial respiration; on the escape of pus natural respiration returned. Next day all symptoms had disappeared, and for seven days patient appeared practically well. Tenth day: temperature 96°, pulse 50, apathetic, screaming fits from pain in the head, pupils dilated, stabile. Acute hydrocephalus diagnosed; chloroform, finger introduced into trephine opening, no new abscess found, descending cornu of lateral ventricle tapped by Keen's method, much fluid escaped under pressure. Following day remission of all symptoms. Report from pathologist, "pus of abscess contains a pure culture of pneumococcus." Fifteenth day: without previous warning the following symptoms rapidly appeared: temperature 105°, pulse 140, wild delirium, unconsciousness, squint, etc. Diagnosis: acute purulent infection of the distended ventricles, which was confirmed on withdrawal of the tiny tube that had been left in the descending cornu, when bubbles of air and purulent cerebro-spinal fluid escaped. An attempt was made at once to irrigate the lateral ventricles from the descending cornu with warm, sterile, normal saline solution, under the expectation that it would escape from the fourth ventricle into the cerebellar abscess, and thence out through the cerebellar trephine opening. Though the cerebellar trephine opening was discharging cerebro-spinal fluid the plan failed, probably because of some valve-like action of the wall of the opening. Nothing now remained but to press the use of anti-pneumococcic serum. Great improvement resulted. In 36 hours the ventricles contained nothing but cerebro-spinal fluid. The wounds assumed the pink colour characteristic of successful antitoxin injection, and ceased to discharge pus; the pulse, temperature, and general condition were greatly ameliorated, and consciousness returned. Six days later marked retraction of the head occurred, and the patient again became unconscious and died. *Autopsy.*—In the cerebellar hemisphere a second abscess was present which had not been opened, though the finger had been introduced several times into the cerebellum and pushed in various directions. The unopened abscess was of considerable extent, antero-posteriorly and transversely, but in depth it scarcely exceeded an eighth of an inch; its upper surface corresponded to the upper surface of the hemisphere, and the sense of resistance to the finger was attributed to the tentorium.

As illustrating the difficulty of palpating the cerebellum in cases of abscess the following case may be mentioned:—Female, age 12 years, had a large cerebellar abscess in anterior part of right lobe. Four days later a second abscess behind the first was evacuated. Symptoms relieved, but in a few days recurred. Cerebellum again explored with the finger; at the upper, inner, and posterior part of the hemisphere a sense of resistance was felt, which was thought to be a thin layer of normal cerebellar tissue intervening between the finger and the tentorium. Death next day. *Autopsy.*—Two abscess cavities had been drained, but a third abscess was present of flattened shape, lying just underneath the tentorium, which had been mistaken for normal cerebellar tissue.

Abscess in unusual situations.—Though abscess beneath the tentorium usually occurs in the substance of the hemisphere it may be met with in other situations.

(*a*) *In the middle lobe of the cerebellum.*—Septic softening may extend from an abscess in the lateral lobe into the middle lobe; or an abscess may form in this lobe independently. The symptoms that may be looked for are extreme cerebellar incoördination, rotatory nystagmus, paresis of the trunk muscles, occipital headache, severe vertigo, and frequent vomiting.

(*b*) *Abscess of the flocculus.*—In addition to the symptoms of cerebellar gait, nystagmus, vomiting, optic neuritis, etc., there was, in a case of my own, implication of the 5th, 6th, 7th, and 8th cranial nerves, and profuse epistaxis.

(*c*) *Abscess in the pons.*—This may occur as a primary affection, or in consequence of inflammatory extension from other parts. The symptoms that may be expected are various paralyses involving both eyes, both sides of the face, and the limbs on both sides. The most common combination is paralysis of the face on one side, and of the limbs on the other—the face being paralysed on the side of the lesion. Other symptoms are difficulty in swallowing, possibly pin-head pupils, and finally death by arrest of respiration.

Diagnosis between brain abscess due to ear disease and certain other diseases.—(*a*) *Tuberculous meningitis and tuberculous tumour.*—The symptoms and duration of tuberculous meningitis vary so greatly that diagnosis is often difficult, especially in childhood. When associated with chronic purulent otorrhœa the disease has been mistaken for brain abscess, and operative treatment undertaken which of course failed in its object. It is important to remember how often otitis in children is tuberculous, and that symptoms of intracranial disease, simulating brain abscess, may arise from the presence of a tuberculous mass or masses in the brain, or from tuberculous meningitis. I have many times experienced this difficulty in diagnosis. The cases of supposed tuberculous meningitis in which suspicion of abscess is likely to arise are those of ear disease with palsy. The salient features in which a case of tuberculous meningitis differs from one of brain abscess are—(i.) the temperature is above normal; (ii.) the pulse is 100, or more rapid; (iii.) optic neuritis is absent or is a late symptom; (iv.) vomiting is neither so urgent nor so frequent as in abscess; and (v.) the child is apathetic from the onset of illness, or, even before illness is suspected, is dull or irritable.

The predominance of certain localising symptoms in cases of tuberculous meningitis, especially of hemiplegia, has long been well known; and, before the treatment of brain abscess by operation as a systematic procedure came into practice, these symptoms were often considered in relation to the diagnosis of meningitis from tumour. Several years ago I operated upon a case in which right hemiplegia was associated with left purulent otorrhœa, under the notion that a temporo-sphenoidal abscess was present; but the case proved to have been one of tuberculous meningitis.

Illustrative cases:—

Case 1.—Female, age 6 years. Ill three weeks with occasional vomiting and right purulent otorrhœa. Mastoid operation had been performed two years before, on this side, for extensive disease. The child lived two weeks. During this time the salient points of the case were: The vomiting did not recur. Temperature was normal, or slightly above normal. Pulse varied from 90 to 120. *Convulsions of the left side occurred.* Both pupils were dilated and fixed. No optic neuritis. Irregular respiration and Cheyne-Stokes breathing were observed towards the end. Head occasionally slightly retracted. Child apathetic and fed with difficulty. The diagnosis was tuberculous meningitis involving chiefly the posterior fossa; at the autopsy this was confirmed, the disease being most marked about the medulla and pons.

Case 2.—(Not under my care.) M., age 12. Right purulent otorrhœa, vomiting, drowsiness, temperature 100°, pulse 110, weakness of left arm, no optic neuritis nor oculo-motor paralysis; family history of consumption. Right temporo-sphenoidal lobe explored for abscess. Autopsy a few days later disclosed tuberculous meningitis.

Case 3.—A young child was under the care of a most distinguished clinician, now deceased, for three months. Left hemiplegia and right ear disease were present. The diagnosis was tuberculous meningitis. *Autopsy.*—Chronic abscess of the temporo-sphenoidal lobe.

Case 4.—Male, age 13 years. Ill for three weeks. When seen, drowsy, restless, and complained of severe pain in the frontal and left temporal regions, no paralysis, no anæsthesia, no tenderness of head, no optic neuritis, knee-jerks and reflexes equal, pupils equal and reacted slowly, pulse 50, temperature 99°, retention of urine, respiration slow, head sometimes retracted, had vomited several times, drums normal, upper posterior quadrant on each side exhibiting merely a slightly more pink colour than normal; strong family history of consumption. Diagnosis: tuberculous meningitis with acute hydrocephalus. Ordered three leeches over each mastoid, great relief from pain; next day more conscious, headache much relieved, but condition otherwise unaltered. Lumbar theca tapped, not sufficient cerebro-spinal fluid escaped to indicate distension of the ventricles, but mind became clearer and questions were answered; pulse went up, after operation, to 120, but temperature was subnormal; this clearly showing that the condition from which the boy was suffering was not acute hydrocephalus. Nothing further was done, and a few days later the patient died. *Autopsy.*—Large left temporo-sphenoidal abscess, drums intact and normal, muco-pus in both tympana, no caries, some greenish discoloration of dura over left tegmen tympani.

(*b*) *Marantic thrombosis of the sinuses.*—In young children this thrombosis as a complication of marasmus is not uncommon, and is sometimes associated with ear disease and paralysis. The main facts which in these cases distinguish them from abscess are: (i.) The temperature above normal; (ii.) the pulse more rapid; (iii.) the slight degree of ear disease; and (iv.) the alternating paralysis of the eyes and face.

Case 1.—Infant, 1¼ years. Ill three weeks with wasting and vomiting. When seen, temperature 100°, pulse 120, left otorrhœa, right facial palsy of cerebral type, no optic neuritis, champing movements of jaws, fontanelle full, no head-retraction, said to have had an alternating squint. A week later, *left* facial palsy, but no right facial palsy. *Autopsy.*—Buffy, non-septic clot in left lateral, straight, and superior longitudinal sinuses; vein behind each ascending parietal convolution plugged with clot, that on the left side most extensively so.

Case 2.—Infant with right otorrhœa and weakness of left upper extremity which a week later cleared up, and weakness of the opposite upper extremity appeared. P.M. much the same as in Case 1.

(*c*) *Localised suppurative meningitis.*—The diagnosis of this condition from cerebral abscess presents sometimes almost insuperable difficulties.

Illustrative cases:—

Case 1.—Female, age 49. Profuse left otorrhœa seven years. When seen, completely comatose; had been three weeks ill. Symptoms during the illness: severe headache, monosyllabic aphasia, paralysis of left third nerve, left pupil dilated, stabile, left optic neuritis, right hemiplegia, right partial hemianæsthesia; several convulsions had occurred; temperature, pulse, and respiration had been throughout illness above normal. Left temporo-sphenoidal lobe explored; from one puncture about half a drachm of thick yellow pus was obtained. *Autopsy* (next day).—A thick layer of yellow pus was found exactly covering the area of the left frontal and parietal lobes. Extensive disease of left tympanum, no temporo-sphenoidal abscess.

The high temperature, the rapid pulse and respiration, together with the convulsions, pointed clearly to the invasion of the cortex rather than to the presence of an intra-cerebral abscess.

Case 2.—Female, age 7 years. History: said to have had double otorrhœa for a long time, measles four months ago; family history of phthisis. When seen had been ill three weeks, severe frontal headache alternating with drowsiness; no optic neuritis, nor ocular paralysis, had had several fits, temperature 101·5°, pulse 100, respiration 20, pus found in left meatus, but none in right ear, which appeared healed; weakness of left arm had been noticed for three days. Diagnosis: temporo-sphenoidal abscess, tuberculous meningitis or localised suppurative meningitis. A temporo-sphenoidal abscess could not possibly have produced weakness of one arm without involving either the face or leg. Two days later the left arm was reported to be recovering power, while the right arm was becoming paralysed. *Autopsy.*—Suppurative meningitis spreading backwards from both frontal lobes. Origin of meningitis unexplained; frontal sinuses normal.

Case 3.—Male, age 5 years. Operation for extensive right mastoid disease. Three days later, high temperature, quick pulse, no vomiting, weakness of opposite face and arm. Obviously cortical irritation, not intra-cerebral abscess. Temporo-sphenoidal region explored, thick layer of pus on surface, none in lobe. *Autopsy.*—Extensive suppurative meningitis right side.

(*d*) *Embolism, hæmorrhage, and thrombosis.*—When an elderly patient, who happens to have a discharge from the ear, presents symptoms of brain lesion, we naturally inquire whether the brain disease had any connection therewith? In the aged the temporal bones are sclerosed, and if tympanic disease arises it cannot produce an infection of the brain until sufficient time (months or years) has elapsed for the inflammatory process to pass through the dense boundaries of the tympanum; the comparatively rapid intracranial infection seen in young children, with unclosed sutures and porous bone, cannot occur.

Again, in abscess of the brain due to ear disease, the onset of the brain symptoms is gradual, and they may not reach their acme for two or three weeks, while in vascular lesions of the brain the symptoms may be fully developed in a few hours or, at most, days. In embolism the onset is usually instantaneous and unconsciousness is rare. In hæmorrhage the patient may be a sufferer from chronic heart or renal disease; the onset is usually rapid, and arterial pressure in excess. In thrombosis the manifestation of the symptoms is more gradual, and may extend over a few hours or days.

Although no autopsy was obtained, I append one illustrative case—one out of many of the kind I have met with—in order to enforce the point of the difficulty of diagnosis:—

Case.—Male, age 62. Influenza. For three days had pain in left ear and profuse otorrhœa. On fourth day became drowsy, and then gradually comatose. When seen, left hemiplegia, limbs on right side rigid, no vomiting, temperature 102°, pulse 120, eyes turned to left, no optic neuritis. Patient had never had ear disease before this attack. The age, the short history of otorrhœa, the absence of vomiting, the condition of the pulse and temperature, and the onset of coma in the course of a single day, excluded the diagnosis of brain abscess, and made the diagnosis of thrombosis almost certain; a diagnosis easily explained by the weakening influence of influenza.

Some general remarks.—Although, when in a patient who is suffering from chronic purulent otorrhœa symptoms of brain abscess appear, it might be thought quite obvious that the infection had arisen from the temporal bone, yet cases do arise which seem to show that this is not always so. In one case, after severe influenza, the patient died with abscess in the occipital lobe; in another, two months after a severe attack of the same disease, the patient died with an abscess in the frontal lobe. Both had chronic purulent otorrhœa. This is not the place to describe the surgical technique of the treatment of these abscesses, but it may be pointed out that no exploration for abscess should be looked upon as having failed until the finger has been introduced along the track of the trocar and canula, and determined the absence of a tense abnormal swelling. It is by no means uncommon to have a return of symptoms a few days after the evacuation of abscess, due either to the refilling of the abscess from faulty drainage, or to the formation of a new abscess in another part of the same lobe. In the cerebellum it is by no means infrequent to have a second or even a third abscess. After the drainage of a brain abscess, whatever the symptoms of further cerebral trouble that may arise, and they are often inexplicable, the duty of the medical attendant is to explore the brain through the original wound, and not to be led away to undertake a chance operation in another region. Instead of concentrating the attention on the original site of abscess, the new symptoms—such as high temperature, rapid irregular pulse, screaming fits, retraction of head, general twitchings, vomiting, drowsiness, etc.—may suggest conditions which are not present, such as meningitis or acute distension of the ventricles; the symptoms being really due to the refilling of the old abscess or to the formation of another. The temperature, pulse, and respiration of brain abscess are modified when the skull is opened, in consequence of the relief of intracranial pressure. There is great danger in delaying operation when a brain abscess has been diagnosed, especially in the case of abscess of the cerebellum, since death may occur suddenly from pressure on the respiratory centres. Should the natural breathing cease during operation, artificial respiration must be carried on while the operation is continued, and the cerebellar abscess opened; pressure is thus relieved, and natural breathing restored. It has been thought that in cases of double otorrhœa with symptoms of abscess of brain a difficulty would arise in determining the side of the abscess. It has always happened, in my experience, that the tympanic disease showed signs of greater activity on the side which determined the intracranial infection; although in cases of double otorrhœa without intracranial complication it is not uncommon to find the lesions and evidences of activity practically symmetrical.

It is remarkable that after operation and apparent convalescence some cases of brain abscess begin to retrograde and finally end fatally. These are cases in which large areas of the brain are involved, and the healing process fails at a certain stage. A similar event occurs occasionally after the removal of large brain tumours, and depends on a general nutritional failure.

Illustrative Case.—Male, age 38. Discharge from left ear eleven years, complete left facial palsy seven years, vertigo, pain in head, and sallow complexion. Mastoid operation, small abscess size of Barcelona nut found in petrous; two or three days later he asked the nurse to boil a sixpence (he had previously been in the habit of giving her sixpence to buy eggs with). When seen next day, aphasia, agraphia, and alexia, vomiting, right face and arm slight weakness, right knee-jerk increased, temp. 97°, pulse 60. Operation: whole left temporo-sphenoidal lobe turned into abscess. Abscess wall of old standing; wound healed well, but feeble state of patient unequal to repairing so great a cavity in the brain, and death occurred in the eighth week. Autopsy showed healing process nearly complete; the abscess had been plugged from the bottom with gauze for five weeks.

II. **Diffuse suppurative meningitis.**—The symptoms of localised suppurative meningitis, which subsequently, of course, may become diffused, have already been discussed under the diagnosis of brain abscess. When the inflammation is diffused the headache is very severe, the patient often crying out with pain; optic neuritis occurs early; there is rigidity of neck with some implication of the cranial nerves, such as that causing squint or irregular respiration; the pulse is full and rapid; the temperature high without the great oscillations of pyæmia; vomiting occurs, and paralysis of the limbs according to the greater or less amount of invasion of the motor cortex.

Occasionally a case is seen in which very high fever and pain in the ear suddenly set in, the patient having previously seemed in perfect health, with the exception of slight chronic otorrhœa. Severe headache and (usually) vomiting soon occur, followed by unconsciousness, and in two or three days by death, the medical man, in these meningitis maligna cases, having scarcely time to complete his diagnosis or to determine whether surgical interference be necessary or possible; all this is very different in the cases of brain abscess in which the onset is much more gradual.

Illustrative cases:—

Case 1.—Male, age 30, was seen late one evening. Patient had gone to his work in the morning perfectly well; he had had otorrhœa for two years. The acute illness began suddenly without warning at 3 P.M. with agonising headache; temp. when seen 105·5°, face pale, lips blue, breathing rapid; he had been crying out with pain in the head until unconsciousness supervened. Already he had incipient optic neuritis; nothing in chest to account for the condition. Diagnosis: meningitis. The patient lived for three days. *Autopsy.*—Suppurative meningitis most marked over the base, but spreading over the vertex: tympanum extensively carious on the side from which there had been otorrhœa. From the dura over the tegmen tympani a tiny thrombosed vein passed to the pia mater. It is clear that the pyogenetic infection had proceeded along this tiny vein, and had been the cause of an acute and fierce explosion of suppurative meningitis.

Case 2.—Male, age 35. Double otorrhœa one year. Severe pain in head eight days, with shivering, restlessness, and delirium. March 25th: very stupid and torpid. March 26th: very restless and could not be kept in bed, hence brought to hospital. On admission; double otorrhœa, œdema over left mastoid, severe pain left side of head, looked very ill, temp. 101·6°, no optic neuritis, no paresis. March 27th, 2 A.M.: rigor, temp. 105·4°; 4 A.M., operation on left side, large antrum full of stinking pus, tegmen antri and tympani gone, subdural abscess over anterior surface of petrous. March 28th: extremely restless, temp. 105·4°, horizontal nystagmus, conjugate deviation of both eyes to right, slight right facial spasm and palsy, left knee-jerk brisker than right. Operation: sinus explored, no pus found in groove, but sinus contained non-septic thrombus; left cerebellar hemisphere normal, as would be expected with high temperature, absence of vomiting, and the palsy of right side of face, notwithstanding the nystagmus and the conjugate deviation of the eyes to the right. March 29th: died, temp. 106°. *Autopsy.*—Subdural cavity over roof of left tympanum had been drained, dura softened in one place and showed small perforation. Suppurative meningitis was spreading from the temporo-sphenoidal to the frontal-parietal region.

Note the absence of vomiting during the whole illness, the high temperature, and the almost uncontrollable restlessness.

III. Lateral sinus pyæmia.—Occurs both in children and in adults. When tympanic caries is present in childhood the immature state of the temporal bone is favourable to the extension of the septic process to more dangerous regions, to the escape externally of purulent collections, and to earlier and clearer indications of deep-seated disease than are commonly met with in the adult. At every age lateral sinus pyæmia is fatal, unless early operative interference be carried out. In twenty-two out of fifty-seven necropsies on ear cases a purulent thrombus was found in the lateral sinus. It is important to note that pyæmia from disease of the temporal bone may prove fatal without the occurrence of thrombosis of the lateral sinus.

Symptoms.—(i.) Those due to the local inflammation. A purulent discharge from the ear and a history of its presence for more than one year. The carious process must have time to extend from the antrum or tympanum to the bony groove for the lateral sinus. A subdural collection of fœtid pus is usually found in the bony groove, which, by its presence next the dura mater bounding the sinus, inflames its wall and infects its contents. Local œdema and tenderness along the course of the sinus and of the internal jugular vein. Stiffness of the muscles of the back and side of the neck, sometimes causing retraction of the head. Pain in the ear.

(ii.) Those due to general infection of pyæmia. Sudden onset with pain in ear, frontal and occipital headache, shivering, vomiting, and oscillating temperature. Rigors are repeated day by day as in pyæmia. Vomiting may recur several times, but in pyæmia is not a prominent symptom, as it is in brain abscess. The temperature runs up to 105°, and down again perhaps to normal—one or two such oscillations taking place every twenty-four hours. The mental state may remain unimpaired up to the time of death.

Early diagnosis.—In a certain proportion of cases there is no pain in the ear and no otorrhœa; thus the attention may not be drawn to the ear, and the examination of it may be omitted. Years ago I saw an autopsy on a patient, supposed to have died from typhoid fever during a typhoid epidemic, in whom the lesions of lateral sinus pyæmia were found.

In young children a diagnosis has to be made without rigors, though sometimes convulsions take the place of rigors. The oscillating temperature indicates the septic state. In children, as compared with adults, the temperature easily runs up; and, unless the general state of the child correspond to the temperature, and be one of serious illness, too much stress must not be laid upon it. It must be remembered also that though the septic process is usually of pyæmic character, it may resemble septicæmia and be fatal as such without rigors; both in children and adults. The diagnosis is often obscured by the application of leeches and blisters, which inflame the soft parts over the mastoid and prevent the proper examination of the region. As a rule the otorrhœa is horribly offensive, but sometimes it is slight and inodorous; yet this inodorous discharge may be as serious as one which is offensive, for the organisms giving rise to pyæmia do not necessarily give an offensive odour to the pus. There may be no local œdema over the mastoid, in fact no masto-squamous abscess; because the long-continued, deep-seated inflammation has sclerosed the bone around the antro-tympanal cavities. Tenderness may exist only at the posterior border of the mastoid, and is a local sign of deep-seated collections of pus in the bone or sigmoid groove. In one case, in which there was great swelling of the soft parts over the lower part of the mastoid and upper part of the neck, offensive pus was found escaping from the mastoid foramen and mastoid vein; and this was subsequently proved to have come respectively from a subdural abscess in the groove, and from a suppurating clot in the sinus. The swelling and tenderness in the neck in the course of the internal jugular vein may be due to phlebitis of the wall of the vein, which is distended with clot; or the vein may be empty and collapsed, and swelling will then be attributable to enlarged and inflamed lymphatic glands. Optic neuritis may be present in cases of lateral sinus pyæmia; if present it means that the dura is so far invaded as to allow of the spread of a basal meningitis to the region of the optic nerves. The presence of optic neuritis adds to the urgency of immediate operation.

Diagnosis from typhoid.—The sudden onset, the oscillating temperature, the repeated rigors, make the diagnosis of lateral sinus pyæmia usually easy; but otorrhœa may supervene in the earliest stages of typhoid, and in patients who have otorrhœa enteric fever may begin with rigors. Murchison says: "Enteric fever would be excluded from the diagnosis by a temperature approaching to normal on any evening during the first week, and on the other hand by a temperature of 104° on the first day, or second morning of illness"; and again, "3 out of 63 cases of enteric fever commenced with rigors: in several instances, not included in this analysis, I have observed decided rigors, in fact all the phenomena of ague, during the first few days." This experience shows that the occurrence of rigors and even a sudden onset of illness do not exclude typhoid, and that the diagnosis of lateral sinus pyæmia must not rest only on a series of rigors. Clearly, if optic neuritis is present, early typhoid fever is excluded; and the absence of knee-jerks favours a diagnosis of intracranial inflammation rather than of enteric fever.

Illustrative cases :—

Case 1.—Male, age 20. When seen, headache, earache, vomiting, and general malaise; six days previously had had a rigor, and onset of illness was therefore sudden; discharge of fœtid pus from meatus last six months, temperature 104°. During next few days temperature was continuously high, and he had no further rigor; on seventh day a typical typhoid eruption appeared and cleared up the diagnosis. If the temperature had oscillated to normal, or if a series of rigors had occurred, the lateral sinus would certainly have been examined by operation.

Case 2.—Female, age 12. Discharge from left ear seven years. Repeated shivering two days. When seen, severe headache, tongue furred, temperature high, fœtid pus escaping from left auditory meatus. The further history of this case was much like that of the other; there was little daily variation of the temperature, no further rigor occurred, and a typhoid rash came out.

Whether the sinus be full of clot, or of moving blood, it matters not, as far as treatment is concerned. In either case the poison of pyæmia is being poured into the sinus, or, say, through the vein of the cochlea into the jugular; and the same method of treatment must be adopted. When there is no septic thrombosis the artificial thrombosis produced by the true surgical treatment may erect an effectual barrier against further immediate infection from the primary focus of disease. The average duration of untreated lateral sinus pyæmia is three weeks.

The intracranial complications that are commonly met with are suppurative meningitis, brain abscess, and extension of the thrombosis to other cerebral sinuses. In one case the patient, a woman, aged 30, suffered from melancholia as a complication of the pyæmia, and pushed a carpet needle into her heart, which was not discovered until the necropsy.

Notwithstanding evidence of secondary deposits the cases, though desperate, are not without hope; and the medical attendant should advise surgical treatment, not only for the primary disease, but also for the accessible secondary purulent foci.

Pneumococcus infection.—A group of symptoms similar to those which arise in septic infection of the lateral sinus may occur from pneumococcus infection. Without wishing to dogmatise, it may be stated that the pneumococcus causes lesions in many other parts of the body besides the lung, some of which are suppurative and others undergo resolution. There is reason to suppose, both on clinical and bacteriological grounds, that a general febrile disorder, quite comparable to other specific fevers, is sometimes induced by the pneumococcus, which may or may not be accompanied by local inflammatory changes in the lung or elsewhere. The middle ear is one of the most frequent extra-pulmonary seats of the pneumococcus; and the otitis thus caused may occur independently of any recognisable pneumonia, or may precede an attack, or follow it. In one of the cases of brain abscess referred to in this paper (p. 591) the pus yielded a pure culture of the pneumococcus. Joint suppuration is also caused by the pneumococcus, and it is probable that in many cases in which there is acute pain in one or more joints we have really to do with a pneumococcus inflammation, which undergoes resolution. In some of the cases in which intracranial suppurative lesions, such as meningitis, follow acute otitis with unusual rapidity, particularly when accompanied by marked joint pains, this explanation of general pneumococcus infection with multiple localisations may be thought of.

Cases illustrative of pneumococcus infection:—

Case 1.—Male, age 7 years. High fever, no evident cause; two days, then headache and earache, right tympanic membrane red and bulging. Pus evacuated, it yielded almost pure culture of pneumococcus. Fifth day: pneumonia right base. Recovery perfect.

Case 2.—Three members of one family were taken ill in succession; the first had a febrile attack with joint pains, the second had suppurative otitis, and the third had a severe attack of pneumonia. All three made good recoveries.

Case 3.—*Recovery from severe meningitis with otitis, probable pneumococcus infection.*—Male, age 7 years. Sudden onset, with vomiting and headache, on May 10th. Seen May 12th: pulse 108, temperature 101°, drowsy and moved with difficulty, tongue coated and brown, no history of phthisis; next day wild delirium, some retraction of head, no paralysis, right optic disc blurred, screaming, convulsions, convergent squint. Temperature continued high, but with large daily oscillation for next fortnight, but kept below 100° from May 27th to June 15th, when there was a temporary renewal of fever. Optic neuritis became well marked, but the squint only lasted a few days; some days after admission purulent discharge from right ear, but without relief to the symptoms. Frequent convulsions occurred. After several months' illness the patient, though he reached the last degree of emaciation, ultimately recovered, improvement beginning on July 17th. He was discharged from hospital on September 23rd in fair health, and without much impairment of hearing.

Cases illustrating lateral sinus pyæmia and some fatal complications:—

Case 1.—*Ambulatory lateral sinus pyæmia.*—Male, age 27. Travelled sixty miles, and walked into the writer's room. Right fœtid otorrhœa fifteen years, earache and shivering eight days, since then shivering, sweating, headache, vertigo, temperature 104°. Operation same day. No swelling over mastoid, large stinking cholesteatomatous cavity in mastoid surrounded by hardened bone. Suppurating clot in sinus, upper segment of divided vein stitched to skin of neck. Rapid and complete recovery.

Case 2.—*Lateral sinus pyæmia operated on too late.*—Female, age 40. Fœtid left otorrhœa twenty years. When seen had had a rigor every day for three and a half weeks; temperature varied in rigors from 105° to 107°. Condition unrecognised because there was neither tenderness nor swelling over the mastoid, but for several days there had been swelling all down the left side of the neck.

Mind clear, slight icteric tinge of skin, pulse rapid and feeble. Operation: immense cholesteatomatous cavity in mastoid surrounded by eburnated bone. Suppurating clot in sinus and in vein as far as subclavian. After operation temperature normal; death occurred on fourth day, apparently from heart failure.

Case 3.—*Pyæmia complicated by albuminuria in an alcoholic subject.*—Male, age 36, publican. Six months before had had influenza followed by profuse discharge from right ear. When seen, fœtid discharge from right ear, temperature 104°, rigors, vertigo, vomiting, no mastoid signs, albumin one-sixth. Operation: large suppurating cavity in mastoid, much pus in groove, disintegrating clot in sinus. Vein divided between two ligatures, upper ligature taken off. Next day temperature normal, urine contained albumin and blood. A week later died. Post mortem: large white kidneys.

Case 4.—*Lateral sinus pyæmia, cerebellar abscess, death from extension to opposite lateral sinus.*—F., age 14. Right otorrhœa seven years. Earache and rigors commenced three weeks ago. For one week rigors have ceased, and there have been drowsiness, photophobia, normal temperature, and vertigo. Operation: inner and outer walls of sinus sloughed, torcular end had to be plugged, cerebellar abscess evacuated. Death four days later. *Autopsy.*—Purulent thrombus between torcular and plug. There was an abnormally free communication with the opposite lateral sinus, and the clot had extended across the middle line into the opposite lateral sinus.

Note.—Never plug the torcular end of the sinus if it can possibly be helped; for the plug may dam up septic material which may cause extension of the thrombus to the opposite lateral sinus; or, what is more usual, to the straight or superior longitudinal sinus, with which the left and right lateral sinuses are respectively continuous. If a plug be necessary it should be removed as soon as possible.

Case 5.—*Lateral sinus pyæmia, extension of septic thrombosis.*—Male, age 22. Right otorrhœa seven months, when typical symptoms of lateral sinus pyæmia supervened. Operation: internal jugular vein divided in neck, upper end sutured to skin incision. Lateral sinus opened behind ear; proximal end had to be plugged with gauze, as it bled freely. For twenty days the patient went on well, but pulse was slow, bowels constipated, and skin very sallow. On twenty-fifth day, as temperature had risen and patient had been several times sick, and a little offensive pus had escaped from the wound behind the ear and from the opening of the jugular vein on the surface of the neck, the mastoid region was explored. Venous hæmorrhage occurred from the superior petrosal sinus, which was difficult to stop. For a few days the condition improved, but then the mental state became altered, and he was generally sick once a day; temperature and pulse were above normal. In reading he made many mistakes, and frequently wept; the right arm was weaker than the left, and there was slight paresis of the left side of the face. The question arose whether the condition were due to further extension of the thrombosis or to the development of a brain abscess. The temperature, which was of an oscillating character, reaching 102° daily, and the bilateral paralysis were in favour of thrombosis. A circle of bone was removed over the temporo-sphenoidal lobe, but the dura was not opened, as there was no evidence of intra-dural distension. The same evening he had a fit of tetanic type, beginning on the right side and afterwards spreading to the left; the head and eyes were turned to the left, and as the fits spread to the left side they were turned to the right; the fits were frequently repeated, lasted seven or eight minutes, and during them fæces and urine were passed involuntarily. *Autopsy.*—Superior longitudinal sinus filled with a puriform clot, as was also the left lateral sinus, which had been infected by a small commissural vein crossing the torcular from the right lateral sinus. The veins passing from the parietal lobes to the superior sinus were plugged with clot, and there was here some yellowish exudation over the convexity of the hemispheres ; a plugged vein on each side about an inch behind the furrow of Rolando was peculiarly prominent. The right superior petrosal sinus was not plugged, and it was from this that the bleeding had occurred, as the right lateral sinus seemed firmly plugged with clot. In the left hemisphere, immediately below the upper end of the Rolandic furrow, was a small recent abscess like an almond in outline and size.

CASE 6.—*Lateral sinus pyæmia, extension of thrombosis to cavernous sinus.*—Male, age 8 years. Admitted with right otorrhœa, temperature 105°, rigors and all the symptoms of pyæmia. Immediate operation : vein divided between two ligatures, upper ligature not taken off as vein bled freely. For one week patient was practically well, when temperature rose to 103°, pulse 120, respiration 24 ; was sick, but not drowsy, and there was no optic neuritis. Next day right eye prominent, ecchymosis of conjunctiva, ophthalmoplegia interna. Next day left eye similarly affected. Twenty-four hours later death. *Autopsy.*—Right lateral sinus and upper segment of internal jugular vein contained pus which had overflown into the inferior petrosal sinus.

Note.—Never leave a ligature on the upper segment of the vein for more than a few hours, otherwise the vein may become a test-tube of pus which will infect the cavernous sinus. Septic thrombosis of the cavernous sinus is inevitably fatal, but the writer sees no reason why such a condition may not be successfully dealt with by operation, provided, of course, that interference takes place before the septic clot has crossed the middle line by the transverse and circular sinuses.

CASE 7.—*Lateral sinus pyæmia, extension into superior longitudinal sinus.*—Female, age 14 (not under care of writer). Typical symptoms : right lateral sinus involved ; usual operation. Case progressed well for a week, then temperature gradually rose to 103°, and vomiting, headache, screaming fits, drowsiness, and paresis of right face and arm occurred. Right cerebellar hemisphere explored, nothing found. *Autopsy.*—Right lateral and superior longitudinal sinuses contained purulent thrombi. Opening in sinus plugged with clot, behind which was offensive pus. The tributary veins going to superior longitudinal sinus from left motor cortex were thrombosed, and over the left motor cortex was slight suppurative meningitis.

IV. Lateral sinus septicæmia.

Illustrative cases :—

CASE 1.—*Acute septicæmia following otitis media occurring during convalescence from scarlet fever.*—Female, age 6 years. Acute otorrhœa with swelling and œdema over mastoid. Operation : membrane incised, mastoid opened. Temperature normal first three days ; when seen again a week later, temperature 104°, respiration 30, pulse 140, jaundice, diarrhœa, enlargement of liver and spleen, distension of belly, albuminuria. General infection had occurred. It was useless to attempt by operation to prevent further infection from the primary focus. Ordered 10 c.c. anti-streptococcic serum every six hours, altogether 260 c.c. injected. Within twenty-four hours it was clear that the serum was controlling the septic process, and seven days after the first injection temperature was normal. Large hæmorrhages occurred into the areolar tissue in various situations, but this hæmophilic sequel of the septic process was successfully dealt with by antiscorbutic diet and the administration of calcium chloride. Complete recovery took place.

Case 2.—Male, age 5 years. Scarlet fever followed by right acute otitis, temperature 103°, knocking pain in ear. Operation: incision of membrane and opening of antrum. Improvement for two days; then temperature rose gradually, and in a week had reached 104°. Pulse was rapid and irregular, no signs in lungs. Mastoid explored found to be cellular and to contain pus; disease removed, convalescence rapid.

Note.—The mastoid in children may be as pneumatic or diploetic as in adults,—contrary to what is usually stated.

Case 3.—*Lateral sinus septicæmia, no diagnosis made.*—Male, age 2 years. September 3rd: operation for double hare-lip. September 10th: lip healed satisfactorily. September 19th: some breaking down of wound, and child looked ill and refused food. September 21st: lip wounds completely broken down; from this time to October 6th, when he died, there was gradual emaciation, temperature occasionally 99° but usually normal; towards the end some petechial spots appeared on the skin. Sepsis was suspected, but its origin could not be determined. *Autopsy.*—Pus in right tympanum, which was carious, membrana tympani normal, puriform clot in right lateral sinus, small hæmorrhages in the pia, pleuræ, and lungs.

Note.—The absence of signs of inflammation over the mastoid is no proof of the absence of extensive disease in the tympano-antral cavities, but in these cases in infants there is usually profuse external otorrhœa. Note also the intact tympanic membrane with a carious tympanum, and the absence of rigors and high temperature.

V. Lateral sinus sapræmia.

Female, age 30; 5½ weeks before seen had been confined. Three days after parturition temperature 105°, uterus curetted, no effect on temperature, which rose every day to 105° or 106°; there was no shivering nor vomiting. On the tenth day uterus again curetted; meanwhile the husband and one servant had been taken ill with typical enteric fever, and the question arose, Was this also a case of typhoid? When seen by the writer temperature had been 105° or 106° every day for 5½ weeks. A day or two before, some deafness of the right ear had been discovered. Mind clear, no optic neuritis, patient thin and weak, tongue furred, temperature 106°, no recollection of a discharge from the ear, though on right side drum was absent, and on wiping promontory with a little cotton wool the wool returned slightly damp and offensive. Operation same evening: antrum deeply placed, surrounded by eburnated bone, and about twice the size of a pea, full of granulation tissue; no pus. Result: no rise of temperature after operation, rapid convalescence.

Charles A. Ballance.

CEREBRAL HÆMORRHAGE

For the purposes of the present article the title may be taken to include intracranial hæmorrhages in any position. There is no part of the contents of the cranium which can be said to be free from the liability to rupture of blood-vessels, but of course some parts are more prone to hæmorrhage than others.

That a diseased condition of the arterial walls is a necessary condition seems certain; yet there are many cases, those, for instance, occurring in apparently healthy young persons, in which such a condition has not been demonstrated.

Causation.—*Remote Causes.*—Persons of certain physical conformation have long been regarded as especially liable to cerebral hæmorrhage; such as have a short, thick neck, a florid complexion, and so forth: but experience teaches that the lesion is at least as common, if not more so, in persons of quite opposite build. Undoubtedly, however, there are certain general conditions which dispose to rupture of cerebral vessels.

First among these must come that state in which are associated, more or less, interstitial fibrosis of the kidneys, hypertrophy of the left ventricle, and other evidences of a high arterial blood-pressure. Whatever the causal relation of these three factors to one another, there can be no doubt that in such cases there is a strong tendency to degeneration of the cerebral arteries; unfortunately, however, in the present state of knowledge, the extent of such degeneration must be one of inference only. Perhaps in the greater number of cases the only hint of such a condition during life is the raised arterial pressure, as evidenced by tortuous radial and temporal arteries, incompressible pulse, accentuated aortic second sound, and perhaps some evidence of hypertrophy of the left ventricle. Such patients may or may not present a history of the arthritic phenomena of gout, and, after death uratic deposits may be found in the cartilages of their joints. In 50 necropsies in cerebral hæmorrhage at St. Bartholomew's Hospital—in which the great toe and other joints were opened—uratic deposits were found in 19 instances (38 per cent); in the remaining 31 the cartilages were quite normal. There can be no doubt that the part played by gout is a very important one, yet the arthritic aspect of the matter is perhaps the least important.

There is a striking tendency to heredity in cerebral hæmorrhage. A single lady, aged 41, during a violent attack of sea-sickness, became unconscious, and died in the hospital: a large hæmorrhage was found, in the usual position, which had burst into the lateral ventricles. There was no atheroma of the cerebral arteries, and but very little of the aorta. The kidneys were but slightly affected, and the heart scarcely at all hypertrophied. But nine members of her family, who during life had presented little or no evidence of arterial or kidney disease, had died in the same way.

Certain poisons dispose to cerebral hæmorrhage: of these the most important are syphilis, lead, and perhaps alcohol. Whether alcohol may operate as a direct cause is not decided; more usually its influence is indirect, by inducing a tendency to degeneration, an influence which may affect the offspring.

In the later degenerative stages of syphilis the cerebral vessels seem to share in the general degradation of tissue, but in some instances the brunt of this degradation may be said to fall upon these vessels; it is possible that this may explain some of those cases in which extreme cerebral arterial change is found without corresponding heart and kidney signs.

Persons exposed to the influence of lead are, as is well known, particularly prone to gout; and it is probably through the mediation of gout that the liability in such persons to cerebral hæmorrhage arises.

Certain general blood states dispose to hæmorrhage, and in these the brain tends to suffer equally with other tissues: such are pernicious anæmia, leucocythæmia, purpura, and scurvy.

Age.—No period of life is exempt from cerebral hæmorrhage, but inasmuch as arterial degeneration is most common in the later periods, it is after middle age that we find the chief liability to such hæmorrhage. On this point, however, there seems to be a want of agreement in the published statistics. The following table has been prepared from the post-mortem records of St. Bartholomew's Hospital during eleven years. In that period 3790 necropsies (medical) were made, including 132 cases of intracranial hæmorrhage.

The age was ascertained in 124 cases:—

	Males.	Females.	Total.	Per cent.
8 - 20 years . .	6	2	8	6·4
21 - 30 ,, . .	4	3	7	5·6
31 - 40 ,, . .	11	6	17	13·7
41 - 50 ,, . .	32	11	43	34·6
51 - 60 ,, . .	18	6	24	19·3
61 - 70 ,, . .	14	6	20	16·1
71 - 80 ,, . .	5	0	5	4·0
	90	34	124	

From this it will be seen that the liability seems to be greatest between 40 and 50, and to decline appreciably with each succeeding decade. These figures correspond in the main with those of Fagge, made from a similar source to the above.

Gintrac's figures have been freely quoted by succeeding authors. They have the value of large numbers, but they are largely made up of recorded cases, and are therefore to a large extent selected. According to Gintrac's tables the percentage of cases increases with each decade from the age of 30, to attain the maxium between 61 and 70. It is also noticeable that as many as 17 per cent fall between 70 and 80. This estimate, then, does not accord with the experience of two of the large London hospitals.

It is possible that senile degenerative processes may in some cases fall more particularly upon the arterial system, a circumstance which will explain the rupture of vessels in old people; but it is contended that an inherited or acquired tendency to arterial degeneration will lead to a dangerous condition of the vascular wall long before old age.

Sex.—As to sex there can be no doubt that women are much less liable to cerebral hæmorrhage than men. Here perhaps the statistics of the post-mortem room of a general hospital may lead to an erroneous conclusion. Reference to the above table shows 90 males (72·5 per cent), and 34 females (27·4 per cent). Many of the men were brought in from the street or from their work, where they had fallen; women, who would be more likely to be seized at home, would be more likely to remain at home. Perhaps Gintrac's conclusions are nearer the truth; namely, that the relation between male and female cases is about 57 to 43 per cent.

Although women are equally liable with men to the hereditary causes, they are less likely than men to acquire the arterial degeneration.

It seems to be accepted almost as an axiom that cold weather disposes to cerebral hæmorrhage; to test this point the following table was drawn up:—

December	9	March	11	June	14	September	2
January	10	April	10	July	9	October	20
February	13	May	10	August	12	November	10
	32		31		35		32

It is true that the figures are few, but the agreement in numbers between the seasons is rather unexpected. Gintrac's figures sustain the impression that the accident is more likely to happen in the cold months.

An appreciable percentage of cases occur in the course of *infective endocarditis*. In the present series there are 6 only, out of 132; but it is probable that this figure rather understates the true percentage. In this case the accident usually occurs in persons under middle life.

The circumstances under which hæmorrhage may take place are very various. It is quite obvious that if a vessel wall is so weakened as to be on the point of rupture, any sudden rise of blood-pressure (or acceleration of heart-beat) may precipitate such an accident. Hence we find cerebral hæmorrhage occurring under every possible form of excitement or strain, physical or mental—as, for instance, in whooping-cough, defæcation, stooping or lifting, vomiting, coitus, or sudden mental excitement. Many of the cases brought to the hospitals have begun under the preliminary exaltation of drink. Yet in many instances the rupture takes place under circumstances of complete quiescence, though on this point figures are wanting. A certain number of attacks occur during sleep. Of 246 hemiplegics, in 47 (19·1 per cent) the onset was in sleep. It is, of course, impossible to say how many of these were due to hæmorrhage, and how many to thrombosis; in fact, it is probable that a large number were thrombotic. Though the general circulation is quieter in sleeping than in waking, and though it is probable that the cortex in sleep is anæmic, it does not follow that there is any great diminution of the blood-pressure in the main cerebral system of vessels; indeed, Spehl arrives at the conclusion that, though the brain as a whole contains less blood during sleep than in waking hours, certain parts of the base (the ganglia ?) are probably in a state of relative congestion.

General anatomical and physical considerations.—The copious blood-supply necessary for the cerebral functions is brought to the base of the brain by four large vessels, which here form the remarkable anastomosis known as the circle, or polygon, of Willis. The anterior or carotid element of this is perhaps the most important in the present connection.

The internal carotid may be regarded in three divisions. The anterior division, called the anterior cerebral, passes forwards and upwards over the genu of the corpus callosum to supply the inner aspect of the hemisphere and the greater part of the anterior lobe. It anastomoses with its fellow by the anterior communicating artery, and it gives off branches internally to the head of the nucleus caudatus and to the corona radiata of the anterior lobe. The middle division, the middle cerebral or Sylvian artery, is practically the direct continuation of the internal carotid; it is a large vessel, but its calibre is much smaller than that of the carotid, and its diminution in size is almost sudden. It is the most important of the cerebral vessels; it courses laterally over the anterior perforated space into the fissure of Sylvius, to be distributed to the greater part of the anterior lobe, and to all the parietal region, including therefore the Rolandic area and a portion of the posterior cortex. In passing across the anterior perforated space it gives off a number of branches at right angles to its own axis, which pass in to supply the so-called basic ganglia, the internal capsule, and part of the optic thalamus. One especially large and important branch passes between the external capsule and nucleus lenticularis, and then into the latter; and this branch has been shown by Charcot to be particularly prone to disease and rupture; the third division, if so it may be called, is the posterior communicating, which passes backwards to join the posterior cerebrals, thus completing the circle of Willis. Under normal circumstances this is a small vessel; but it is an important one, as it forms the basic anastomoses between the carotid and vertebral systems.

The vertebral arteries on entering the cranium give off the posterior cerebellars, and then join to form the basilar artery, which runs forward, grooving the pons Varolii, and giving off many branches, laterally and vertically, to supply the pons and cranial nerve nuclei. Close to the origin of the basilar arise the median cerebellars distributed to the inferior aspect of the cerebellum; and at the anterior edge of the pons are given off the superior cerebellars which supply the superior aspect of the cerebellum.

The basilar divides a little in front of this into the two great posterior cerebrals, which course round the crura cerebri to supply the cortex of the temporo-sphenoidal and posterior lobes, sending in branches to nourish the crus, optic thalamus, and corona radiata of the posterior lobes. The anatomical disposition of the vessels in the brain has been exhaustively treated of in the works of Duret.

The dura mater is supplied by (i.) the anterior meningeal, derived from the anterior ethmoidal branch of the ophthalmic artery; (ii.) a few twigs from the ascending pharyngeal; (iii.) the middle meningeal, from the internal maxillary, a large vessel which supplies practically the whole of the dura mater except the extreme anterior part and that of the posterior fossæ covering the cerebellum; (iv.) the small meningeal, a branch of the last to the Gasserian ganglion and dura mater of the middle fossa; (v.) the posterior meningeal from the vertebral, supplying the dura mater of the posterior fossæ.

Besides the basic anastomosis of the circle of Willis there is every reason to believe that there is a free anastomosis between the cortical cerebral vessels. Duret admits the occasional existence of an anastomosis, but considers it to be less free and constant than other writers. However, Heubner and others, Biscons, Mendel, and Tedeschi seem to have demonstrated this anastomosis conclusively. The branches to the nuclei of the base, however, are terminal. This point is illustrated by a case in which the middle cerebral was found completely plugged soon after its origin from the carotid; the caudate and lenticular nuclei were so softened that they could be almost shelled out, while the nutrition of the cortex was everywhere perfect. If therefore, as has been suggested (Tedeschi), there is any anastomosis between basic and cortical branches, it must be very inconsiderable.

Peculiarities of the cerebral circulation.—The following features of the cerebral circulation may explain, to some extent, the liability of the vessels to disease.

(*a*) The brain receives its blood by two distinct channels, the carotid and vertebral.

(*b*) In comparing these two systems it may be noted that the diminution in size is more gradual in the vertebral than in the carotid; which may explain the greater liability of the latter to atheroma and rupture.

(*c*) The part of the organ requiring the greatest blood-supply is outside, therefore the main divisions of the great vessels are distributed over the cortex by gradual dichotomous division, comparable with the usual division of arteries all over the body. It must be owing to this circumstance that hæmorrhage is less common by far in these vessels than in those to the internal parts.

(*d*) The vessels for internal distribution come off, as above described, at right angles from the main trunks; such as the middle cerebral, posterior cerebral, basilar. This means friction, for the less the angle made by two branches the less the resistance to the flow; but these vessels are very small, they have a comparatively short course, and are practically terminal. Indeed, as Mendel suggests, it is probable that the pressure in these small vessels is not very much less than that in the carotid itself. The strain then upon the walls of these vessels is probably far in excess of that on the walls of vessels of a corresponding calibre in the cortex or elsewhere in the body.

(*e*) By the researches of Bayliss and Hill doubt has been thrown upon the existence of a vaso-motor mechanism in the brain. They obtained no evidence of such a mechanism by stimulating the vaso-motor centre, the central end of the divided spinal cord, or the stellate ganglion. According to these writers, the cerebral circulation follows passively the changes in general arterial and venous pressure. Gulland (5), Morison (31*a*), and others, however, have found nerve fibrils on the vessels of the pia mater which may be vaso-motor. It is difficult, moreover, to accept the proposition that an important system of vessels, provided with well-developed muscular coats and supplying blood to such a highly-specialised organ as the brain, should be unprovided with any special direct controlling mechanism whatever.

Pathology and Morbid anatomy.—(A) The state of the blood-vessel. (B) Associated lesions elsewhere. (C) Mechanical effects of hæmorrhage. (D) Situation of the focus of hæmorrhage. (E) Secondary degenerations.

(A) *The state of the blood-vessel.*—It is obvious that before rupture of a vessel can take place there must be some preparatory weakening of its wall, and of the causes of such a weakening the most important is atheroma.

Atheroma generally manifests itself in the form of patches of yellow thickening of the internal coat of the vessels, showing distinctly through the thin walls. These patches may be of considerable size, and may affect arteries of all sizes from the carotids and basilar to small arterioles of less than a millimetre in diameter. The condition is supposed to be preceded by thickening of the internal coat, of an inflammatory nature at first—an endarteritis. This thickening may remain as such, with a tendency to increase, and so to obliterate the lumen of the vessel, at the same time causing an atrophy of the middle or muscular coat, a condition resembling the lesion known as endarteritis obliterans which occurs in syphilitic subjects. The effect of this is to stiffen the wall, and to dispose rather to thrombosis than to rupture.

But in other cases, and in the majority, this thickening tends to caseous degeneration and softening, so producing the characteristic yellow patches above referred to. The change may stop here, and again tend to favour thrombosis; or the caseation may proceed to the deposit of lime salts with great impairment to the elasticity of the wall, though not necessarily so weakening it as to lead to rupture. Such a state of affairs, as Charcot and Bouchard have suggested, may however tend to raise the pressure in the small arterioles beyond, and so to aid in the formation of miliary aneurysms in these arterioles. But the caseation frequently takes a different course; it softens and breaks down; and then there results an actual weakening of the wall with, sooner or later, the formation of an aneurysmal dilatation, the bulging of which is favoured to some extent by the comparative want of support of the cerebral vessels by surrounding tissues. These aneurysms show some similarity in size and appearance, and perhaps also in some of the conditions of their formation, to those found in the lungs in phthisis. They vary in size from that of a pin's head to that of a pea, and sometimes much larger—as large as a small chestnut in one case (Bastian, 2). They are found most commonly on the middle cerebral, or its large branches; and frequently in the fork formed by the division of the vessel. Out of 108 cases of persons dead of cerebral hæmorrhage, they were found in 18; and of these 11 were on the middle cerebral. It should be stated, however, that Gull found aneurysm of the basilar more common than of any other artery, thus:—basilar, 20; middle cerebral, 15. These aneurysms are frequently multiple, and no doubt will account, by their rupture, for many of the arachnoid hæmorrhages; though they are often impossible to find on account of the large quantity of surrounding clot. It must not be supposed, however, that aneurysms of this size are common causes of intra-cerebral hæmorrhage. Atheroma of the cerebral vessels is found in an overwhelming percentage of cases of hæmorrhage. Out of 108 cases in which this point was definitely noted, more or less atheroma was found in 92 (84·2 per cent).

Conversely, however, atheroma is a fairly common lesion, whether associated with cerebral hæmorrhage or not. Out of 675 necropsies during the last two years the cerebral arteries presented more or less atheroma in 46; and during the same period there occurred 25 cases of cerebral hæmorrhage of which, say, 84 per cent presented atheroma. We may conclude, then, from these figures that atheroma without cerebral hæmorrhage is at least as common again as atheroma with it.

Miliary aneurysms.—In 1868 Charcot and Bouchard (15) made their memorable observations on aneurysms of the smaller arterioles, to which they gave the name miliary aneurysms (*vide* vol. vi. p. 333). These take the form of minute dilatations of the arteriolar wall, sometimes fusiform and elongated, sometimes distinctly saccular. They can be seen with the naked eye, as minute red grains on the vascular twigs. These writers considered the lesion to be essentially a periarteritis. They admitted that atheroma may coexist in the larger vessels, but did not consider that atheroma has any direct relation with these aneurysms or with cerebral hæmorrhage. The part they assign to atheroma is the stiffening and want of elasticity of vessels affected by it, and the consequent transmission of the shock of the heart-beat to vessels less able to bear it and already weakened by this periarteritis.

Subsequent observers have placed the initial lesion in the internal coat. Eichler and others have looked upon it as a combined lesion, a peri-endarteritis. The common-sense view is to look upon atheroma and miliary aneurysm as manifestations of the same process. No doubt miliary aneurysms are found in some cases without atheroma, and the reverse is true; but is it necessary on that account to suppose a different morbid process in the two cases?

The great central fact in the chain of events is the rise of arterial tension or stress on the vascular wall. Hypertrophy of the left ventricle is a part of this process, and thus the abnormal shock of the pulse-wave becomes an accessory factor. As the vascular walls become more and more rigid, the pulse-wave is less and less taken up in its transmission; and it arrives at the small twigs with a force much less modified than is normally the case. Why in some cases there should be miliary aneurysms without apparent intervention of atheroma is a difficulty of the same nature as the frequent want of proportion between the amount of hypertrophy of the left ventricle and the extent of an interstitial fibrosis of the kidney.

The effect of continued increase of blood-pressure upon the vascular wall is admirably illustrated by the state of the pulmonary artery under such circumstances. The pulmonary artery is less often affected by atheroma than the aorta, as it is less subject to increases of blood-pressure. But in any condition which does raise the pressure in the vessel—such as mitral disease or emphysema—yellow specks of atheroma soon appear, distributed chiefly about the bifurcation which receives the full force of the pulse-wave.

For a more detailed account of these arterial lesions, see the article on "Arterial Degenerations and Diseases," vol. vi. p. 319.

Infective endocarditis.—Before leaving the consideration of the vessels, notice should be taken of a not uncommon termination of infective endocarditis; namely, rupture of an embolic aneurysm. Aneurysms of this nature are caused by the blocking of a vessel by an embolus,—usually a small tag of fibrin from the valve,—loaded with microbes. By their proliferation the microbes give rise to an acute inflammation of the wall of the blocked vessel, which results in the rapid formation of an aneurysm, which may rupture. Out of 134 cases of cerebral hæmorrhage 6 were ascertained to be of this nature; but it is probable that this estimate is considerably under the true proportion (*vide* art. "Embolism," vol. vi. p. 251).

Aneurysms of the cerebral vessels occurring in young persons are generally of this description. Dr. Church, some time before the microbic origin of such aneurysms was understood, showed that they are generally associated with diseases of the valves of the heart; and he gives a list of 13 cases, all between the ages of 13 and 20.

(B) *Associated lesions of heart, kidney, and general circulation.*—The correlation of these factors, and their relative importance in cerebral hæmorrhage, are matters about which there is still much diversity of opinion. An attempt has been made in the following table to arrange these lesions so as to show the frequency of their association. The figures are made up from a total number of 110 cases of cerebral hæmorrhage:—

Associated Lesions.	Males.	Females.	Total.	Percentage.
1. Atheroma of cerebrals and aorta. Hypertrophy of left ventricle. Chronic interstitial nephritis.	55	17	74	66·5
2. Atheroma of cerebrals and aorta. No hypertrophy of left ventricle. Chronic interstitial nephritis.	4	2	6	5·4
3. Atheroma of cerebrals and aorta. Hypertrophy of heart. No disease of kidney.	4	4	8	7·2
4. Atheroma of cerebrals and aorta only.	4	2	6	5·4
5. No atheroma. Heart hypertropied. Chronic interstitial nephritis.	4	2	6	5·4
6. Chronic interstitial nephritis only.	2	2	4	3·6
7. No lesion of vessels, heart, or kidney found.	2	1	3	2·7
8. Aneurysm of cerebral vessels without any other lesion.	5	0	5	4·5

It will be seen at once how commonly granular kidney, hypertrophy of the left ventricle and atheroma are found in association. Atheroma may exist alone, or without one or other of the usually associated lesions, as shown by Nos. 4, 2, and 3. On the other hand, we find no atheroma in 5, 6, and 7, making a total of 13 cases, or 11·8 per cent.

Great stress has properly been laid upon the evidences of chronic high tension or stress of the coats of the arteries, and its probable causal relation to arterial degeneration; but we still find a considerable percentage of cases of cerebral hæmorrhage without such evidences as above shown. According to Charcot and Bouchard, and other Continental observers, this percentage is still higher. To explain such cases it seems almost necessary to look for some inherent tendency to arterial degeneration; especially in those cases, which occur occasionally in the experience of every physician, in which the rupture takes place in young and apparently healthy persons who presented no evidence of high blood-pressure either before death or after. In this same category must be included that form characterised by early death and a tendency to run in families.

Nothnagel draws attention to the observation of Frerichs that out of 241 cases of Bright's disease, 6 only suffered from cerebral hæmorrhage; that is, 2 per cent. This observation is not borne out by experience at a London hospital. In order to test this point it is found that, during the last two years, out of 675 necropsies there have been 116 cases of all diseases in which the kidneys have been more or less after the granular type; and this number included 20 out of 25 cases of cerebral hæmorrhage, or 17·2 per cent; thus the coexistence of the two conditions is much higher in this country apparently than on the Continent.

(C) *Mechanical effects of hæmorrhage.*—When a considerable vessel bursts in the soft yielding brain-substance there is formed at first a more or less oval focus, with its long axis parallel to the line of least resistance; that is, in the length of the fibres if the rupture be in the white matter. If it is in the common position between the nucleus lenticularis and the external capsule, it will tend to separate these two structures. If the leak be a gradual one the effused blood may clot quickly, and so stop any further leaking, for a time if not permanently. In this case the clot gradually changes colour, turns brown, softens, and slowly becomes absorbed. The cerebral substance round the focus is generally stained, sometimes for a distance of a quarter of an inch. This stain is at first a claret red, but soon alters to brown, and finally to yellow. At the same time the lacerated brain substance undergoes fatty change, and eventually disappears; then a cavity is formed, often with a distinct cyst wall (Charcot, 12), containing a yellowish fluid, with at one period masses of brown amorphous pigment in it, or even crystals of hæmatoidin. Where the extravasation of blood is but small, absorption is so complete that only a yellow stain may be left behind—the so-called ochreous patches.

But very frequently the effusion of blood is too rapid for such a favourable course. The blood is then pumped into the soft brain tissue, and with each beat of the heart its power for destruction and laceration increases, on the principle of the hydraulic press, by virtue of which, as the cavity enlarges, the power exerted by the blood-pressure and pulse becomes enormously multiplied:[1] in fact it is difficult to see how a hæmorrhage into the brain ever ceases, unless by pressure on the artery itself.

The brain itself now becomes compressed so that on opening the cranium the convolutions are found to be flattened; at first chiefly on the side of the hæmorrhage, and later on both sides.

The advance of the destructive hæmorrhage is practically always towards the centre; so that, unless stopped, rupture into the ventricles follows sooner or later. The lateral ventricle on the same side is first filled with blood, which then oozes across through the foramen of Monro into the opposite ventricle, and downwards into the third ventricle so as to distend the infundibulum, which, being thin, may rupture, and blood appear in the arachnoid space about the base. At this stage the corpus callosum will be found to bulge upwards, and to fluctuate. The fornix and lower aspect of the corpus callosum may be softened and eroded. The blood may find its way down the descending cornu of the lateral ventricle and reappear at the base; but this passage is generally stopped by compression. In hæmorrhages of this size the blood will often be found along the aqueduct of Sylvius and in the fourth ventricle, from which it may find its way upwards and round the peduncles of the cerebellum, coating the latter with a thin layer of clot.

Rupture of vessels of the base gives rise to an effusion of blood beneath the arachnoid. In such hæmorrhages the arachnoid cavity of the base will be filled with clot which, owing to its entanglement with the number-less fine fibrils of the arachnoid, is generally very firm; it may also spread up from the base so as to form a thin coat of clot over the whole brain.

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[1] This power can be rather strikingly demonstrated by directing a stream of water at a very low pressure, say 1 mm. of mercury, into a thin indiarubber air-ball. Even at this low pressure the ball will inevitably burst, after distension to its utmost. If a small piece of the same rubber be tied over the mouth of the delivery tube it will sustain a great pressure of mercury, say 100 mm. or more.

Hæmorrhages into the cortex rarely open externally (Charcot, 13), but follow the general rule of thrusting towards the centre. When, however, they do rupture externally, the blood tends to spread all over the surface in the arachnoid space.

(D) *Situation of the focus of hæmorrhage.*—By far the commonest seat of hæmorrhage is in the region of the lenticular nucleus; and, as before mentioned, the ruptured vessel is most commonly that branch of the middle cerebral which runs between that nucleus and the external capsule. However, in the larger number of cases the destruction of tissue is so extensive as to render the discovery of the primary vascular lesion impossible; yet the centre of the mass of clot may be taken, approximately, as the site of the initial lesion; and in the following cases this point has been taken as the guide to its situation.

Out of 118 cases in which the seat was noted we find 76 (64·4 per cent) in which the focus lay external to the lenticular nucleus and the anterior part of the optic thalamus; of these, 32 were on the right side and 44 on the left. The pons Varolii is the next and most common situation; in 19 cases the lesion was primary and in the pons only; in 11 others there were small hæmorrhages in the pons, but the main lesion elsewhere. So that in respect of vulnerability of the vessels of the pons the figures should stand at 30 (25·4 per cent).

The corona radiata, or white matter of the frontal region, comes next, but with a considerable drop in the numbers. The vessels here concerned will probably be the anterior cerebral, or again the deep branches of the middle cerebral. Of these we find 6 only, 5 of which were on the left side.

Foci confined more to the optic thalamus, and due to rupture of branches of the posterior cerebral, occurred in 5 instances only, of which 4 were on the right side.

The corona radiata of the posterior lobe was the seat of hæmorrhage in 3 cases.

The cerebellum presented foci of hæmorrhage in 2 cases only; Sir W. Gowers says this hæmorrhage most frequently follows rupture of a branch of the superior cerebellar to the dentate nucleus. The crus cerebri was affected in one case, but a large hæmorrhage in the thalamic region may not infrequently extend backwards down the crus of the same side.

In comparing the liability to rupture of the two systems of vessels—the carotid and vertebral—we find that the accident is just twice as frequently in the former as in the latter; namely, 84 of the carotid system, and 41 of the vertebral (the 11 pons cases included above come on both sides of the account).

Hæmorrhage into the lateral ventricles is rarely primary. It was believed to be so in 2 of the cases. According to Duret, the walls of the ventricles are mostly supplied by the posterior cerebrals. The choroid plexus receives blood from the carotid as well as from the vertebral system.

Miliary aneurysms have been found on the vessels of the choroid plexus. But secondary hæmorrhage into the ventricles is a very common incident, as may be judged by the fact that it was found in 56 of the above cases (47·4 per cent).

Lastly, hæmorrhage may take place into the arachnoid space, and this occurred in 4 instances, in which the ruptured vessel was not found. This form of hæmorrhage is generally named meningeal hæmorrhage; but obviously it is due to lesion of the cerebral rather than of the meningeal system of vessels.

True meningeal hæmorrhage, due to rupture of branches of the meningeal arteries, is most often traumatic. The effusion of blood may be on either side of the dura mater,—that is, extra dural, or subdural. Subdural hæmorrhages of considerable size may occur in some blood diseases; such as purpura, scurvy, and pernicious anæmia. The extravasated blood forms a thin layer of clot over the hemisphere, sometimes on both sides symmetrically. The clot is sometimes laminated, the various layers indicating definite extravasations; the most recent being next the dura mater. Moreover, Dr. Herringham has shown that the hæmorrhage can actually be seen in the substance of the dura mater, in the form of ecchymoses sometimes of considerable size.

Subdural hæmorrhage, however, is more often seen in asylum practice; especially in cases of general paralytic dementia. An inflammatory origin of this clot has been maintained by Virchow, a view which seems to be still accepted abroad. In this country, however, the simple hæmorrhage hypothesis obtains generally (Wynne, Wigglesworth). Dr. Mott has very kindly given me some figures upon this point, from the records of the London Asylum at Claybury. They are as follows:—Of 114 post-mortem examinations upon male cases he finds 52 (45·6 per cent) were cases of general paralytic dementia; of these, 11 (21·15 per cent) showed signs of cerebral hæmorrhage; in 8 of them the hæmorrhage was subdural only, in 2 of them in the brain substance, and 1 in both situations. The average age of these patients was 41·7 years.

Hæmorrhage is no uncommon accident in the course of the growth of an intracranial tumour, more especially in those highly vascular growths the angio-sarcomata. The hæmorrhage may so plough up the growth, and alter appearance, as at the necropsy to mask the original disease.

The traumatic hæmorrhagic lesions sustained at birth are generally meningeal or subarachnoid (*vide* art. "Cerebral Palsies of Infancy," p. 738).

(E) *Secondary degenerations.*—If the hæmorrhage do not prove fatal, yet the destruction of tissue is complete and involves the motor fibres, and there will follow secondary degeneration of the pyramidal fibres in the medulla and cord. The great bulk of the pyramidal fibres cross, at the decussation of the pyramids, to become the crossed pyramidal tract in the lateral column of the cord. A considerable number, however, do not cross, but run down in the anterior column of the cord next the anterior fissure. A number of uncrossed fibres are found in the "mixed zone" of Flechsig, that is, in the neighbourhood of the anterior roots. All these fibres degenerate completely, and finally become absorbed and disappear, leaving at first a mass of disorganised myeline; their place is ultimately taken by fibrous or sclerotic tissue, which eventually contracts into a patch of scar tissue.

Symptoms.—In this article we are mostly concerned with such symptoms as can be referred to extravasations of blood into or upon the brain, and therefore with the symptoms peculiar to the earlier or acute stages of the attack. The chronic or enduring symptoms, being common both to hæmorrhage and vascular occlusion, are fully treated of under the latter head.

Prodroma, in the ordinary sense of the word, can scarcely be said to exist. Any premonitory symptoms must be attributed to circulatory disturbance, or to small and gradual hæmorrhage; in the latter case such symptoms are practically part of the attack. Atheroma is the commonest cause of vascular occlusion, and it is possible that some of the symptoms called prodromal may be due to it. With this reservation we may mention, among the so-called prodromal symptoms of the older writers, giddiness or dizziness, mental irritability, numbness or tingling of the extremities, and headache. Nothnagel remarks on the importance of a sudden loss of the power of speech, as suddenly regained after a few hours.

The onset of the attack may be sudden or gradual; with or without loss of consciousness. A sudden and complete loss of consciousness is implied in the name "apoplexy." Apoplexy has become so associated with hæmorrhage into the brain that sudden hæmorrhages in other parts of the body are called also apoplectic; notably those in the lungs. Nevertheless the general opinion is that the symptoms of hæmorrhage into the brain are more often gradual than sudden, and that the term "apoplectic" applies less often to hæmorrhage than to embolism (Trousseau). It is, however, usual to speak, somewhat loosely, of the early comatose stage as the apoplectic stage.

The explanation of the apoplectic onset is still rather obscure. It appears to bear an uncertain relation to the amount of blood extravasated; for, as Nothnagel argues, a great hæmorrhage may present a gradual development of symptoms, while a small one may exhibit the picture of apoplexy. The terms "cerebral surprise" (Trousseau) and "cerebral shock" (Hughlings Jackson) serve to convey to the mind the idea of the suddenness rather than of the extent of the lesion. Whether the loss of consciousness is to be referred to sudden rise of intracranial pressure, as the experiments of Pagenstecher would seem to prove, or to interference with blood-supply (anæmia), again in its turn due to pressure on vessels (Abercrombie), or to a condition allied to concussion, must at present be a matter of opinion. Astley Cooper produced a temporary loss of consciousness by pressure of the finger on the brain through a trephine hole; and Bergmann rendered a child comatose by compressing a meningocele: but neither of these authors say how long the loss of consciousness lasted under pressure, or whether consciousness returned without relief of pressure.

The onset may be quite painless; but a sudden sharp pain in the head is not uncommon (Abercrombie), especially when the extravasation is into the arachnoid space: such a pain, followed immediately by coma, suggests also a large hæmorrhage into the ventricles.

Convulsions, generally speaking, are not common. When they occur they usually appear in the early stage. Where they are local they suggest pressure on cortical areas, and therefore extravasation into the arachnoid space. They are also common in hæmorrhage into the pons.

The patient may recover consciousness, partially or completely; or the loss of consciousness of the initial attack may deepen, from a state in which he can be partially aroused, into the deepest coma or carus, which may last for many hours or sometimes for days before death ("Ingravescent apoplexy").

At this period the skin may be found moist with sweat, sometimes so excessively as to saturate the clothes. The veins of the face and neck may be turgid, and the countenance generally cyanosed, but this state will vary with the amount of respiratory embarrassment. On the other hand, in the gravest cases the countenance may be placid and pale, or of natural colour, as in sleep.

The breathing is often stertorous, with noisy rattlings in the throat, due to accumulations within the trachea of secretions, and often of food and drink; the natural and imperious reflex cough being diminished, in common with the other reflexes, by the depth of the coma.

Owing to the complete muscular "resolution" the cheeks will be flaccid, and may be drawn in and puffed out by the respiratory movements. A similar condition of the soft palate may give rise to loud snoring and stertor.

The respiratory rhythm is frequently unaltered, but any alteration is a symptom of grave import. It may be irregular or deep and slow. The Cheyne-Stokes rhythm is generally of fatal augury.

The state of the pulse is variable; more commonly it is slow and deliberate, sometimes it is rapid. An irregular pulse is a sign of grave importance. High blood-pressure is rather a sign of the conditions which favoured the hæmorrhage than a consequence of it. The affection of respiration depends largely upon intracranial pressure, as has been shown by the experiments of Spencer and Horsley, who find that a rise of intracranial pressure is followed by slowing of the respiration and of the heart, even to complete temporary arrest.

Vomiting may occur early in the attack. It is, as Sir William Gowers remarks, especially common in hæmorrhage into the cerebellum.

The position and movements of the eyes and head often give important evidence of the unilateral situation of the lesion. There is a tendency, frequently well marked, for the head to turn to one side; and with this may be seen a conjugate deviation of the eyes in the same direction. This seems to be due rather to unopposed action of the muscles on the side of the lesion than to definite spasm. The patient is then said to look towards the lesion. The importance of this sign was insisted upon exciting cause is apparent, but movement and disturbance are likely to induce it. It is remarkable how often persistent recumbency seems to favour its subsidence. Certainly it often occurs after taking food, and it is influenced to some degree by diet. Ophthalmoscopic examination sometimes excites it.

Constipation is a marked symptom, and usually persists throughout the illness. As a rule it is easily overcome by simple aperients or enemas. *Occasionally it is so obstinate as to secure exclusive attention, and to mask the diagnosis. Thus in two cases of this kind the combination of* obstinate constipation, vomiting, and a slight visible peristalsis raised the *suspicion of intestinal obstruction, and the proposal of laparotomy was for a short time entertained. But in both patients there was marked* ethargy, although no other definite cerebral symptom had appeared.

Rarely there is diarrhœa, which may or may not be associated with tuberculous ulceration of the bowel. In either case the abdomen generally becomes flattened or carinated; the few exceptions being mostly cases of antecedent tuberculous peritonitis. Associated with this carination there s often marked flaccidity of the abdominal wall. The occasional appearance of visible partial peristalsis of the bowels has been mentioned already.

Even at this early stage there is often some inertia of micturition, eading to retention of urine; this can generally be overcome by getting the patient either to stand or sit on the side of the bed.

The *pulse* early shows irregularity, a symptom present with such frequency as to have considerable diagnostic value; the irregularity may affect rate, rhythm or force. The rate per minute alters distinctly during the course of the disease; there may be a little quickening during the prodromal stage, then slowing takes place during the irritative stage, and with the slowing the artery gives a sensation to the touch as if tightly contracted. Marked pulsation of the carotids in the neck may be noticed at the same time; and if the fontanelle be open it is tense and its pulsation manifest. The slowing of the pulse is less marked in young children than in children of five years old and upwards. Finally in the paralytic stage the pulse becomes softer, more and more rapid, and may reach 220 per minute or become uncountable; generally at this stage the irregularity is lost; and in stages of relaxation a few hours before death the fontanelle often becomes flattened again.

Respiration shows a corresponding irregularity, quite early; even sometimes in the prodromal stage an occasional deep inspiration is followed by a deep and long expiration—the so-called "sighing respiration." As the disease advances this becomes more frequent, and towards the end Cheyne-Stokes breathing is commonly seen. Finally the respiration becomes simply shallow and rapid.

A symptom often present, but indicative rather of mischief than of tuberculous meningitis, is *quasi-purposive movements* of the mouth, such as sucking, pursing or biting the lips, champing of the jaw, and grinding of the teeth. Quasi-purposive movements of both upper and lower limbs are by no means uncommon. The repetition of these movements, with slow alternations, may continue for a considerable time, even against resistance. Plucking at the lips, the nipples, and genitals is often present quite early in the disease.

Slight *stiffness of the neck* and even slight *retraction of the head* may be present occasionally, but are very rarely pronounced, or continuous. In the absence of permanent head retraction tuberculous meningitis usually shows a marked contrast to simple posterior basic meningitis. There are, however, very rare cases of tuberculous meningitis, in which head-retraction is pronounced and prolonged, and in which after death an excessive amount of miliary deposit is found at the posterior arachnoid bridge.

Tremor of the head and limbs on movement, associated with rigidity of the neck, is almost always present when the disease is established; and even in an early stage it can generally be elicited if the observer place his hand behind the child's shoulder and raise him slightly from the bed. It is also well seen if the patient attempt to stand, which he does in an unstable fashion with his legs widely separated; his head, arms, and lips showing meanwhile a coarse tremor.

The *convulsions* of tuberculous meningitis may be roughly divided into:—(*a*) The convulsion of invasion already mentioned, which may be general and solitary, and removed by a considerable interval from those which follow; (*b*) the convulsions of the middle period, which tend to be more or less limited; and (*c*) those of the later period, which are frequently generalised. Those of the middle period are the most important and distinctive, because they point to localised cortical disturbance. A varying tonic stage is often succeeded by clonic spasm, limited either to one side of the face and body, or sometimes to one limb. But, although the spasms may on one day be confined to a single limb, on successive days other limbs generally become affected. This is the rule in children, and it points to bilateral distribution of the lesions. Occasionally in adults the spasm is tolerably constant in its limitation, and this is in harmony with the anatomical fact that in adults the meningeal deposit is often more circumscribed than in children.

Sooner or later some *rigidity* of the limbs appears; most commonly it persists after a convulsion, and it often varies on successive days. All four limbs may be rigidly extended, or an arm or a leg may be rigidly extended or flexed. Attempts to overcome the rigidity may elicit expressions of pain, and sometimes may set up rhythmical contractions of other limbs. Convulsions are sometimes followed by *paralysis* rather than by rigidity; and spastic contraction of the limbs of one side may coexist for varying periods with flaccidity from paralysis of the other side. In most cases there is some paralysis of cranial nerves, as might be expected in a disease chiefly affecting the base of the brain. Oculomotor and facial palsy are seen more frequently than palsy of any other cranial nerve. Squint may be one of the earliest symptoms of the disease; and the patient's first complaint, especially if an adult, may be that he sees double. These cranial nerve palsies, like those of the limbs, are often temporary.

Permanent *palsies*, though much less frequent, arise sometimes with, sometimes without preceding convulsions; and of these hemiplegia, brachial monoplegia, facial and oculo-motor paralysis are the most common.

The *speech alterations* of the prodromal stage have been already referred to; but these by no means persist throughout the illness. For the most part they vary like the alternating convulsions and temporary paralyses. Delay in response, or actual speechlessness are more common in children than aphasia properly so-called.

In the later stages there is great *difficulty in swallowing;* but this varies from day to day in a remarkable manner.

Other disorders of movement are seen at times. Mechanically associated movements may be present: for example, on forcibly flexing one leg on the abdomen the arm of the same side or the leg of the opposite side becomes flexed; and probably analogous to this is the adductor spasm in the opposite thigh produced sometimes in tuberculous meningitis when obtaining the knee-jerks; but it is not peculiar to this disease.

In the early stage there is excessive *hyperæsthesia*, though, on account of the general irritability of the patient, it is difficult to gauge. Nevertheless, in some cases there seem to be areas of increased intensity of the hyperæsthesia; as for example over the vertebral spines. In the later stage it is alleged that some *local anæsthesia* can be made out. It is difficult to prove; but at the end of the disease when the patient is becoming comatose, the loss of conjunctival reflex and the easily abraded cornea would seem to favour the opinion that sensibility is lowered.

During the early stage the *superficial reflexes* are not altered; later Henoch specially notes the disappearance of the cremasteric reflex.

Symptoms referable to the *eyes* may be considered in a group. As to the external muscles, varying squint and ptosis are very common during the irritative and pressure stages. If facial palsy occur, either temporary or permanent, the corresponding eye may remain half open for a time.

Independent movements of the eyeballs are often seen, especially slow movements of one or both eyes from side to side; but rarely definite nystagmus. Jerky movements of the eyeballs, either with or without other clonic spasms, and constituting minor convulsions, are sometimes confounded with nystagmus.

The pupils, which are often slightly contracted in the prodromal and early irritative stages, gradually become dilated and show a curious oscillation when exposed to a bright light. The pupil contracts, but immediately dilates again; and then alternately contracts and dilates. Archambault laid great stress on this oscillation of the pupil in tuberculous meningitis; but it is found in other morbid states likewise. In the last stage the reaction to light is lost. The dilatation becomes more marked and persistent as the ventricular effusion increases. In tuberculous meningitis of the convexity, in which case there is not usually any associated hydrocephalus, the dilatation of the pupils does not occur, or is much less marked than in the common form. There is also during the pressure stage some irregularity of the pupils. It has been observed that in some cases the pupil on the side on which the patient lies is more dilated than the opposite one; and in a case recorded by Sir Samuel Wilks, where there was much hydrocephalus, the dilatation was reversed by reversing the position of the head.

Anæsthesia of the cornea and of the conjunctiva occurs during the last stage. There is much vascularity, and a deposit of muco-pus collects on the conjunctiva; and on the lower margin of the cornea there is frequently to be found a crescent of opacity followed quickly by superficial ulceration.

Ophthalmoscopically the two changes which require consideration in this disease are optic neuritis and choroidal tubercle, to which, respectively, attention was first directed by Clifford Allbutt and Cohnheim.

With regard to *optic neuritis* its indications in the early stage of the disease are often ambiguous; but late in the disease it can generally be detected on both sides, though unequal in amount. Even then it is rarely severe or comparable with that associated with cerebral tumour; but the increase of blurring of edge and of curving of vessels is conclusive to one who has observed the progressive change in a given case. In judging of the progress of the neuritis, Garlick has laid great stress in these cases on the alterations which take place in the apparent lumen of the arteries and veins, and on their relation to one another.

Tubercles of the choroid can be observed occasionally in successive development at various parts of the fundus, beginning as minute round dots which become gradually opaque in their centres, and sometimes confluent at their margins. Tubercles of the choroid thus seen to develop during the course of a brain illness are so far in favour of that illness being tuberculous meningitis. I found that out of 16 consecutive cases of choroidal tubercle 13 showed evidence of tuberculous meningitis; whilst 3 presented tubercle in other organs, but no tubercle in the pia mater. Thus, apart from any other signs, tubercle of the choroid cannot be taken as indicating that there is necessarily a deposit also in the pia mater, but it bears witness to a widespread tuberculosis.

The course of the *temperature* in tuberculous meningitis is difficult to distinguish from that of general tuberculosis; but in general terms it may be stated that the advent of the meningeal affection is associated with a lowering of the previous temperature, and with some departure from the hectic type. This lowering of temperature is sometimes of diagnostic value in the first and second stages of the disease; but it is more noteworthy in children of five years and upwards, and in adults than in young children. During the later stage of tuberculous meningitis the temperature rises, in the majority of cases. For a day or two before death the rise may reach 108°; especially when repeated convulsions have occurred. Less commonly the temperature remains low throughout; it may even become subnormal, especially as death approaches.

The *urine* is generally normal in appearance, though said to be deficient in amount. Not infrequently it contains a trace of albumin, and, rarely, a trace of sugar may be found in a late stage of the disease. Both urine and fæces may be passed under the patient from the time that the disease first declares itself.

The *blood* in tuberculous meningitis has been carefully studied by Dr. Bastian. He finds that the white corpuscles are more numerous than in the normal state, and speedily show signs of great amœboid activity by the development of vacuoles within them, and of numerous projections from their outer surface. Groups of protoplasmic particles of various sizes are also to be seen interspersed amongst the blood corpuscles; as well as here and there a small pigment granule, or an irregular block of pigment of reddish or reddish black colour: the red corpuscles, though they present no distinctive changes, usually run together into irregular masses. Bastian holds that these changes are not met with in typhoid fever, or in the great majority of other cerebral affections.

Consciousness is seldom completely lost until quite late: the earlier condition is rather one of drowsy irritability, the patient can be roused, and is sufficiently conscious to resist attempts to feed him, but when left alone speedily relapses into his semi-conscious condition. Occasionally the condition is one of semi-consciousness to the end; the patient can be roused and may answer questions even an hour or two before death. Rarely he remains quite conscious and takes notice of his surroundings until within a few hours of death, when he rapidly becomes unconscious: such cases are usually those in which on some previous chronic disease tuberculous meningitis has supervened. There are not a few examples in every man's experience of the patient's waking up from a coma, recognising his parents, taking food, and even playing with toys, only to relapse again into fatal unconsciousness.

Morbid anatomy.—Tuberculous meningitis is usually a leptomeningitis; but occasionally a few lentil-shaped white granulations are found on the dura mater, and larger tuberculous caseous masses sometimes implicate the dura secondarily.

The arachnoid of the convexity feels slightly greasy, but presents no other sign of inflammation. The superior longitudinal sinus usually contains a scanty pale clot; the veins over the surface of the hemispheres are not generally distended.

The convolutions are flattened, and, as the result of the ventricular effusion, the sulci are less marked than normal. It is in the subarachnoid space at the base and in the pia mater that the most marked changes take place. In the interpeduncular space, over the chiasma, over the anterior inferior portions of the temporo-sphenoidal lobes, and over the anterior portion of the superior vermiform process of the cerebellum, there is opacity due to inflammatory exudation. This is in some places green in colour, and of tough consistency. The Sylvian fissures are firmly glued. In these adhesions some greenish lymph may be found; but on separating the adhesion they are seen to consist for the most part of masses of gray or grayish white tuberculous nodules, which have become partly confluent. Wherever the pia mater extends, such nodules may be traced; predominantly in the Sylvian fissures, but also along the fissures of Rolando, in diminishing numbers upwards to the vertex. There are often small deposits on the two surfaces of the superior longitudinal fissure; and likewise on the adjacent contiguous surfaces of cerebrum and cerebellum. If the pia mater be traced inwards, granulations may be found in the choroid plexuses of the lateral ventricles; the velum interpositum is often packed with them, and so firmly matted that it is difficult to strip it from the subjacent structures. Such granulations vary in size from the finest "writer's sand" ("tuberculous dust") to confluent groups of an eighth of an inch in diameter; and, except at the base, they are seldom accompanied by inflammatory lymph: the brain substance shows occasionally small areas of capillary hæmorrhage in the gray matter. Definite tracts of yellow softening are sometimes to be found also in the areas supplied by branches of the middle cerebral arteries; this softening is induced by the infiltration of the vessel walls with tubercle and by the thrombosis which completes their obstruction. Besides this localised cortical and subcortical vascular softening, there is a widespread alteration of brain substance. The cerebrum as a whole, in a well-marked case, is soft, and the corpus callosum and white matter above the lateral ventricles may be almost diffluent, even when the ependyma is intact, and there is little or no hydrocephalus; so that the softening cannot be the result of maceration. Microscopic investigation throws some light upon this condition: the deposition of miliary tubercle in the subarachnoid, and along the ingrowing processes of pia mater, not only interferes with the vascular nutrition of the brain substance, but actually invades it, giving rise to extensive cellular infiltration, and causing degeneration of both axis-cylinders and ganglion cells. This seems to justify the modern designation of the disease as a true *meningo-encephalitis.*

Small circumscribed deposits of tubercle are not uncommonly found also embedded in the cortex; these are transition forms to the so-called "scrofulous tumours," or caseous tuberculous masses. Such large caseous masses are found in a fair proportion of cases of tuberculous meningitis. Thus, in the series of 100 examples before referred to, Dr. Still found 15 in which such tumours were present. Around these masses there is not infrequently a local eruption of miliary tubercle in the pia mater, comparable with the miliary tubercle over a caseous mesenteric gland in the peritoneum.

Passing from without inwards we find in typical tuberculous meningitis dilatation of the lateral ventricles, and a considerable excess of fluid; this is rarely purulent in the true sense, but is turbid from the partial breaking down of the roof or sides of the ventricles.

The third and fourth ventricles may present slight dilatation; but in tuberculous meningitis there is no closure of the cerebro-spinal foramen, or gluing down of the membranes in the neighbourhood of the posterior rachnoid cistern: in this respect tuberculous meningitis presents a marked ontrast with non-tuberculous posterior basic meningitis, in which, if life e sufficiently prolonged, complete occlusion often takes place, and onsiderable hydrocephalus supervenes, but with clear fluid and firm walls.

There has been much difference of opinion as to the cause of the hydrocephalus in tuberculous meningitis; some regard it as a dropsy, others as the result of an inflammation. Dr. Bastian is inclined to lay stress on the occasional thrombosis of the veins of Galen, and on the general and extensive venous obstruction. It must be conceded that the absence in this disease of adhesion in the neighbourhood of the foramen of Majendie seems to negative the simple mechanical explanation of interference with the drainage system of the brain. Nevertheless, the fairly constant tuberculous infiltration and matting down of the velum interpositum may play some part in lessening the channel of the iter, and thus diminishing the outflow of the lateral ventricles. It seems possible that the anatomical conditions may be the joint result of inflammation and of dropsy; and that the special softening of the walls may be the outcome of the widespread cerebritis above described.

Circumscribed tuberculous meningitis.—Hitherto we have enumerated the generalised morbid lesions found after death in a well-marked example of the disease, which during life gave the complex of symptoms described in an earlier section; and we see how inadequate is the name tuberculous meningitis as descriptive of these lesions. It is important to know that there are cases of restricted tuberculous deposit in the membranes unaccompanied by the other (and, as I believe) more important lesions before described.

(i.) Cases of rapidly fatal miliary tubercle of the convexity. The duration of the illness may have been but a few days, and in the light of the necropsy it is difficult to see why, with such meagre changes, the case was fatal. The amount of miliary deposit on the convexity may be very small; there is little or no accompanying lymph or hydrocephalus; the brain substance shows no important naked-eye alteration, and there is no basal change.

(ii.) Occasionally in the course of the necropsy in cases of general tuberculosis, circumscribed miliary deposit may be found in the pia mater, and small cortical and subcortical deposits of caseous tubercle without any lymph accompaniment, and likewise without subjacent softening or hydrocephalus. In some of these cases there has been no clinical indication whatever of brain affection. It is important to note, however, that the patients have generally been bedridden, and therefore exempted from many of the occupations and trials by which brain symptoms might have been revealed.

(iii.) In rare cases of old-standing tuberculosis, or of tuberculous meningitis (in its generalised form), there may likewise be found at the necropsy a circumscribed area on the convexity, presenting fibrous cicatricial material associated with some small caseous deposit and some scanty granulations—evidently the result of a long-healed tuberculosis.

Finally, with respect to circumscribed areas of tuberculous deposit in the meninges, as compared with the generalised disease which we call tuberculous meningitis, it may be observed that the former is relatively more common in adults, and the latter in children.

Associated lesions of generalised tuberculous meningitis.—In a large number of cases the spinal cord is affected. Of the cases of tuberculous meningitis in adults, analysed by Seitz, 12 out of 20 showed miliary tubercle of the spinal membranes also. In 22 cases of children who died of tuberculous meningitis Dr. Still found tubercle 15 times, either in the theca or on the surface of the cord. The commonest condition observed was fine miliary tubercle, in scanty amount, on the inner surface of the theca, or on the cord in the lumbar region.

In a considerable number of cases excess of fluid, clear or slightly turbid, is found in the theca; and in many instances this fluid has yielded tubercle bacilli (*vide* section on Treatment, p. 488).

Tubercle, in various stages, coexists in many organs of the body. It appears in some situations to be contemporaneous with that in the membranes of the brain—as, for example, in the choroid, on the peritoneal surface of the liver, in the spleen, and even in the lungs and pleura, where either the finest gray or opaque white recent granulations may be sometimes found. But tubercle of older date is to be found in some part of the body. The most common of the older lesions are caseous deposits in the lymphatic glands, especially in those of the neck, at the root of the lungs, at the bifurcation of the trachea, and in the mesentery; and some of these older lesions are often to be found in a state of softening, with evidence of renewed activity and dissemination of tubercle in their immediate neighbourhood, suggestive of invasion of the blood-current.

Purulent collections in conjunction with what has been called surgical tuberculosis—that is, psoas abscess from caries of the spine, hip-joint disease, caries of the temporal bone, or tuberculous salpingitis—may in certain cases be fairly regarded as foci of infection.

The amount of chronic tuberculous deposit may be exceedingly small, and a few examples have been recorded in which no tuberculous focus was found in the body; but it is fair to ask in such cases whether the examination was sufficiently exhaustive.

Prognosis.—The study of the morbid anatomy of this disease gives us the only reasonable basis on which to discuss the subject of prognosis. If the tuberculous deposition be limited to a part of the surface of the brain, there is a possibility of recovery; but if the disease become generalised, so that, besides the invasion of the pia mater, there is an extensive meningo-encephalitis with or without hydrocephalus, the chance of recovery is practically nil.

There are several cases on record in which there was a history of brain symptoms from which the child recovered, but after an interval of months or years succumbed, either to a fresh outbreak of tubercle in some other part of the body, or to an attack of generalised tuberulous meningitis, when after death the evidence of old cicatrised limited uberculous deposit in the pia arachnoid was found.

The duration of the generalised disease has very wide limits. The difficulty of separating the prodromal stage from the onset of the pronounced disease has already been mentioned. The widest variation is observed between the primary and secondary cases. In the primary cases (that is, in those in which tuberculous meningitis supervenes on a state of apparently fair general nutrition) Dr. Gee's estimate of 23 days, reckoning from the time that the disease becomes decided to the day of death, certainly covers a very large number; yet there are a goodly number of longer duration. I have notes of cases in which the disease was definite for 37 days, for 42 days, and for 63 days respectively.

In the secondary cases (that is, in those in which brain symptoms supervene upon some chronic form of tuberculous disease) the duration is invariably short, and may be not longer than three days.

Vertex cases.—Though it is amongst this group that we must look for the very rare examples of recovery from a circumscribed deposit, yet, as a rule, the duration of the cases is much shorter than that of the common basic generalised type. In these vertex cases the patients often pass rather suddenly into coma, and die within a week.

Diagnosis.—Tuberculous meningitis is a disease which presents such wide differences in its early and late stages that the difficulties of diagnosis also vary according to the period at which an opinion is sought. The disease is much more difficult to detect in an early than in a late stage. Speaking generally, the difficulties at *the outset* in children are concerned with either (A) the distinction of the constitutional symptoms from those of some form of *gastro-intestinal catarrh*, or (B) from those of one of the *exanthems* or of *pneumonia.*

In both (A) and (B) there may be headache, vomiting, sleepiness, restlessness, or convulsion. A decision is often impossible for three or four days; but by that time the characteristic eruption of an exanthem or some pulmonary sign of pneumonia should have appeared. In general terms, however, it may be said that at this early period the temperature becomes suddenly and persistently higher, and the pulse and respiration more frequent in (A) and (B) than in the prodromal stage of tuberculous meningitis. The vomiting, though possibly more violent during one or two days in (A) or (B), does not recur day after day with so little distress as is the case in tuberculous meningitis.

Fulness and pulsation of the fontanelle in young children are warnings of incipient meningitis, though they are not conclusive. In the state of exhaustion with semi-coma following severe infantile diarrhœa, to which Marshall Hall gave the name of *hydrocephaloid disease*, the fontanelle is depressed, whilst the abdomen is commonly distended.

The one exanthem which is most difficult to distinguish from tuberculous meningitis in the early stage is enteric fever, as the rash, which in children is often ill defined, may not appear till the eighth day, and in them is often associated with constipation. But, even if constipated, the typhoid patient has some tympanites as a rule, while in tuberculous meningitis the abdomen is retracted. And in tuberculous meningitis the temperature is much lower, and the irritability is greater than in enteric fever. Dr. Stocker's practical sign is often of great value in emphasising this difference: in typhoid, if the bed-clothes be pulled down the patient takes no notice, or at most shows but a languid interest in what has been done; but in tuberculous meningitis the patient resents the interference, and immediately draws the clothes up again. Lateral decubitus is the rule in tuberculous meningitis, dorsal decubitus in typhoid; and the general attitude in the enteric disease is one of relaxation, whilst in tuberculous meningitis there is a proneness to the curled-up position. Even within the prodromal stage of tuberculous meningitis, if the child be raised from the lying posture, there is often an indication of slight stiffness about the neck; but in enteric fever this does not appear. The pupils in early tuberculous meningitis are contracted; in enteric fever they are commonly dilated. An occasional sigh, and an intermission in a slow pulse, are most important suggestions, even in the prodromal stage of tuberculous meningitis; although suspirious breathing sometimes occurs in enteric fever, this is rare in the first week. Headache is sometimes very severe in early enteric fever, but, as pointed out by Sir William Jenner, when delirium appears in this disease, complaint of the head ceases. In meningitis, on the other hand, even after the delirium has appeared, complaint of the head continues. This distinction is more valuable in adults than in children.

Some of the most perplexing cases are those of children presenting a pyrexia maintained for three or four weeks, constipation, extreme and persistent irritability, great resistance to the administration of food, little or no vomiting, no paralysis, and no marked alteration of respiratory rhythm. The irritability is so marked and prolonged that, in spite of the paucity of other symptoms, these cases are generally regarded through the greater part of their clinical course as examples of erratic tuberculous meningitis. Nevertheless, the absolute and sometimes rather abrupt recovery in about a month's time, with the subsidence of the pyrexia, renders it probable that many such cases are really enteric fever, and their occasional association in a family group (other members of which show definite spots) renders the diagnosis sure. In these cases especially Widal's method of serum diagnosis may prove helpful (vol. ii. p. 1145). Bastian's results in the examination of the blood in tuberculous meningitis have already been described; in a doubtful case they also should be utilised in diagnosis.

In adult life, at puberty, and even occasionally before puberty, there arises not very rarely the difficulty of discriminating tuberculous meningitis from *hysteria*. The most reasonable view seems to be that these symptoms of defective control, when manifested in tuberculous meningitis, are as truly hysterical as when associated with some phases of disseminate sclerosis, and even with cerebral tumour. Emotional manifestation in early tuberculous meningitis may unquestionably be arrested by cold douches and by electrical stimuli, and thus a shallow observer may be led to give the assurance that there is no cause for further anxiety. The only safeguard is to suspend judgment on the chance of the appearance of more definite signs, and to pay special attention to the vomiting, to undue somnolence, or to a dazed condition which, in some of these cases, succeeds the period of excitement and drifts rather rapidly into coma.

The occasional mistake of confounding early tuberculous meningitis in adults with *delirium tremens* or *mental disease* has already been mentioned. Every such case must be judged on its own characters; but the undue complaint of head pain and the occurrence of vomiting rarely fail us as aids to diagnosis. It is important to bear in mind that these patients also, like the pseudo-hysterical patients, are very prone to pass suddenly into a dazed condition and thence into coma.

The other group of diseases from which it is necessary to discriminate tuberculous meningitis comprises many different forms of intracranial organic mischief; and, although for the most part the members of this group are simulated in the middle and late stages of tuberculous meningitis, some of them have to be considered in the early stage.

The various brain complications of *ear disease* require careful exclusion. No doubt in the majority of cases, if the ear have been the primary offender, there will be indications of severe local trouble about the mastoid, tympanum, or the meatus, with high fever and sometimes rigors; but sometimes tuberculous meningitis coexists with otorrhœa, or even with tuberculous caries of the petrous bone. In more than one case, trephining of the skull and exploration of the brain for supposed abscess consequent upon ear disease has been performed in cases which have proved to be tuberculous meningitis. The help which the ophthalmoscope yields is not conclusive, for it has been found that thrombosis of the sinuses consecutive to ear disease may be accompanied by optic neuritis. It is also important to note that although we may fail to identify tuberculous meningitis, ascribing its symptoms to the results of ear trouble, conversely one form of ear trouble is often wrongly diagnosed as tuberculous meningitis. This is *double otitis media*, as it is apt to occur in very young children. It often supervenes on general or pulmonary catarrh, pneumonia, and the like, without any preliminary otorrhœa; and the signs of local ear distress may be nil. The child may be fretful and may scream frequently; it vomits now and then; it has an irregular pyrexia; sometimes a tense fontanelle, and, if not actual head retraction, that early rigidity of the neck, seen on lifting the head from the pillow, which has been mentioned amongst the prodroma of tuberculous meningitis. Definite convulsions may occur, either of rigid tonic or bilateral clonic form. These are amongst the multiform results ascribed to dentition. But it is in the experience of many practitioners that such patients may continue sometimes for three weeks with very considerable emaciation, and that recovery may begin after the appearance of a slight purulent discharge from one or both ears. I have been indebted to my surgical colleagues for early incision of the tympanic membrane on both sides in several of these cases; a little pus has escaped, and in several of them recovery was complete. In others the improvement, if any, was but temporary, and ultimately the necropsy showed non-tuberculous meningitis either of the convexity or, more commonly, of the posterior basic or cerebro-spinal forms. The more detailed discussion of these cases is given in the article on "Posterior-Basic Meningitis." But it may be pointed out here that, as between otitis media and posterior-basic meningitis, the latter may be diagnosed when the head retraction is very pronounced, and still more certainly when opisthotonos occurs. Marked head-retraction, as a persistent condition, is very rare in tuberculous meningitis, whereas in posterior-basic meningitis it is the rule, begins early, and may last for weeks unaccompanied by any other active sign except vomiting. In the rare cases of tuberculous meningitis which present severe and persistent head-retraction an unusual and excessive deposit of tubercle at or near the posterior base is generally found at the necropsy. Retraction of the abdomen, on the other hand, such as occurs in tuberculous meningitis, is not characteristic of the cases of otitis media or posterior-basic meningitis; and the same statement may be made with respect to constipation. The whole series of limited clonic convulsions and temporary paralysis which are characteristic of the second period of tuberculous meningitis are conspicuously absent in the posterior basic non-tuberculous form.

Meningitis of the convexity, as a rule, is more rapidly fatal, presents earlier convulsions, and is less prone to dilated pupils than ordinary tuberculous meningitis; but, as pointed out by Dr. Gee, the clinical discrimination of non-tuberculous from tuberculous meningitis of the convexity is impossible. Of the forms of meningitis due to syphilis, whether acquired or congenital, it may be observed (*a*) that they are exceedingly chronic, and (*b*) that they are apt to be associated with choroiditis disseminata, and with coexisting gummata or vascular disease, which are suggested respectively by persistent definite paralysis of cranial nerves and by persistent hemiplegia.

Thrombosis of cerebral sinuses, whether of the septic variety or of that associated with marasmus, is, in some cases, exceedingly difficult to distinguish from tuberculous meningitis. Headache may be very severe, and if there be extensive small cortical hæmorrhages into the gray matter (which are not uncommon in the marasmic variety), short repeated violent screams may simulate the hydrocephalic cry. Extensive hæmorrhages in the fundus of the eye are occasionally a very important aid to the diagnosis of sinus thrombosis; and indications of local venous engorgement about the eyes and temples ought not to be ignored. In the septic variety a possible source like caries of the temporal bone and, subsequently, the suspicion of pulmonary infarction may assist us; and the history of prolonged exhaustion from chronic diarrhœa, or other slowly anæmiating condition, is of value in helping us to guess at the marasmic variety. (Cf. later article on "Occlusion of Cerebral Vessels.")

Abscess of the brain is equally difficult to distinguish from tuberculous meningitis, especially in adults. The existence of a suppurative focus as a possible source, and the occurrence of rigors, are aids when present. But the close parallelism of the symptoms of tuberculous meningitis suddenly supervening upon phthisis, with those of abscess or septic meningitis supervening on bronchiectasis or chronic empyema, is well recognised in clinical experience. The diagnosis hinges more on the pathological antecedents of the patient than on the evolution of the brain symptoms.

Intracranial tumour, especially a tuberculous mass, is frequently mistaken during one of its acute or advancing phases for tuberculous meningitis. This is not surprising, for examination after death shows that multiple tuberculous tumours have often areas of circumscribed tuberculous meningitis in their neighbourhood. But as between tumour and tuberculous meningitis it may be observed that in the first the symptoms are more prolonged than in the second; and even in the active phases they do not show the changes from day to day which are so characteristic of the latter disease. Moreover, as pointed out by Sir William Gowers, the early occurrence of limb symptoms and the gradual loss of power indicate tumour rather than tuberculous meningitis. The ophthalmoscope gives valuable help. The optic neuritis of tumour is much more profound than that of tuberculous meningitis, and, besides showing considerable swelling and sometimes hæmorrhage, is often severe enough to end in atrophy.

Treatment.—*The preventive treatment* of tuberculous meningitis includes everything which is comprised in good hygiene. Some of the points to which recent investigators have drawn attention have been mentioned in the section on causation; namely, a good milk-supply derived from cows free from tubercle, or the sterilisation of the milk when freedom from tubercle cannot be ensured. To this should be added abundance of good food, especially of assimilable fat, and fresh air and sunshine. In those children and adolescents in whom lymphatic gland activity is strongly marked it is most important to remove as soon as possible every peripheral irritation of skin and mucous membranes, lest chronic glandular enlargement be brought about. Pulmonary and abdominal tuberculosis, and some of the tuberculous diseases of bones and joints, must inevitably leave behind them residual deposits, in glands and elsewhere, which cannot be dealt with surgically, and are apt to give rise to fresh infection. In many such cases all that can be done is to invigorate the patient by securing for him good food and favourable circumstances, so that the tuberculous material may become encapsulated. But with regard to glands in the neck, where in many cases the tuberculous material is circumscribed, there is much to be said for the removal of such potential foci of dissemination. If the active inflammatory process in such glands has taken on an autonomy of its own, the proper surgical treatment of it becomes tolerably simple. But in the ambiguous subacute cases the most assiduous efforts should be made, by change to sea or mountain air, and by the use of both local and constitutional remedies, to reduce the peripheral irritation which originally set up the mischief in the glands. When this has been accomplished, if the glands remain permanently enlarged, and still more if they show indications of disintegration, the safest step is to remove them (vol. iv. p. 599).

Treatment of the disease.—We have seen that it is doubtful whether any case of generalised tuberculous meningitis can end in recovery; but, from circumscribed patches of it, it would seem that recovery is possible. Whether any drug can exert a definite influence upon such local outbreaks is difficult to prove; but in mercury we have a medicine which is a powerful germicide, and which appears to modify certain inflammatory processes. Experience on the whole favours its employment. It may be given either as bichloride (ʒj. doses of liq. hydrarg. bichlor.), or combined with iodide of potassium; or as calomel or gray powder; or best of all as an inunction (ʒj. ung. hydrarg. to be applied on a binder daily).

Blisters, setons, and irritating ointments to the scalp are useless and give needless distress. An ice-bag to the head sometimes relieves irritability in the early stage, but often annoys the patient; in cases of hyperpyrexia it is useful to lower the temperature..

If there be much pain in the head, hot sponging sometimes gives relief, and the application of one or two leeches is occasionally helpful.

Bromides and chloral are indicated in the convulsive stage.

The constipation can generally be relieved by gray powder, small doses of castor oil, and enemas.

Feeding is often difficult throughout. In the early stage there may be active resistance; in the middle stage it may be difficult to rouse the sensorium. As with other features of the disease, swallowing is curiously variable. One day there may be great difficulty in the act, on the next no trouble whatever. In the third stage, however, the swallowing reflex seems to be lost, and there is a risk of liquids passing into the larynx. Nasal feeding and nutrient enemas are useful at intervals throughout the disease.

Surgical treatment.—Koch's original method of tuberculin injection can only be mentioned for its pathological interest. It was proved that after the injection there was great increase of hyperæmia around the tuberculous deposits, and that signs of compression became decidedly more evident, in consequence of the inflammatory reaction. Of the value of the later and more benign preparation introduced by Koch we have as yet no evidence.

Lannelongue [quoted by Bernard, 1895] in one case trephined in four places (occiput and frontal), and attempted to irrigate the base of the brain with a through stream of weak solution of bichloride of mercury, in the hope of thus dealing effectively with the chief sites of tuberculous activity in the Sylvian fissures. There was much hæmorrhage from the occiput, and the result was fatal.

Less heroic attempts have aimed simply at the withdrawal of the fluid, either directly from the lateral ventricle or from the subarachnoid space. The lateral ventricles have been attacked through the frontal lobe by Bergmann; through the temporo-sphenoidal lobe by Poirier, Keen, and Kendal Franks; through the arm centre by Mayo Robson. In many of these operations there has been temporary benefit so far as compression signs were concerned; and sometimes the benefit has lasted for four or five days. But the ultimate result has been discouraging. Mr. Robson's case was the most satisfactory. It was that of a girl, aged 10 years, who had hemiplegia with aphasia and optic neuritis supervening on pain and deafness referred to the left ear with otorrhœa. Trephining was performed over the arm centre, and six drachms of serous fluid were withdrawn. At the time of the record considerable recovery had taken place, but there were still occasional spasms of the right arm. In this case the diagnosis of tuberculous meningitis was admitted to be doubtful.

The withdrawal of fluid from the subarachnoid space has been attempted both in the region of the lumbar spine and at the occiput. The non-closure of the cerebro-spinal foramen, and the absence or scantiness of inflammatory deposit in that situation in tuberculous meningitis, render the free evacuation through puncture of the spinal theca comparatively simple. Four of the early cases were reported by Dr. Wynter from Middlesex Hospital. In two of these the first and second lumbar spines respectively were removed, and also a lamina, before the theca was punctured; but in the other two cases drainage was effected by simple puncture and the use of Southey's trocar and tube. There was partial relief of compression signs, but no permanent benefit. Quincke, Ziemssen, Fürbringer, and others have made extensive trials of this method. Quincke makes his puncture with a Pravaz' syringe between the third and fourth lumbar spines at a spot a few millimetres on one side of the middle line; inserting the needle two centimetres deep in the case of a child, four to six centimetres deep in the case of an adult. He asserts that at this site, namely, between the third and fourth lumbar spines, there is no risk of damaging the cord, as the needle enters at the level of the beginning of the cauda equina. Considerable quantities of fluid can be evacuated; there is no difficulty in repeating the operation several times, and the risks are very small. Quincke claims to have saved two patients, both adults, by this method; but the diagnosis was not conclusive in either.

Fürbringer operated in one case (an adult) in which the fluid gave evidence of tubercle bacilli, as verified by control experiments. The case was regarded as desperate, but there was considerable amelioration after the operation, and six months afterwards his state was recorded as good. Nevertheless, as the result of considerable experience in Germany on this operative procedure, it would appear that therapeutically its value is only palliative; for diagnostic purposes, however, it is of some use, especially for the detection of tubercle bacilli in the fluid withdrawn.

The occipital operation has for its object the drainage of the ventricles by means of the cerebro-spinal foramen, and by the posterior subarachnoid reservoir which is bridged over by the arachnoid extending from the cerebellum to the medulla oblongata. Of the two recorded cases, the first, by Dr. Parkin, was proved after death to be an undoubted example of tuberculous meningitis. The incision was made below the superior curved line of the occiput to the right of the middle line. After the membrane had been opened it was found necessary to pass a curved probe forward along the under surface of the cerebellum into the subarachnoid reservoir and into the fourth ventricle. Two or three ounces of fluid were removed with partial relief of compression symptoms, but the child succumbed sixteen hours after the operation. As in the other cases, before the membranes were incised no brain pulsation was to be seen; but with the escape of a little fluid the pulsation became obvious.

The second patient, a girl aged 5½, whose case was recorded by Ord and Waterhouse, made a good recovery, and is an encouraging exception to the somewhat barren results already chronicled. The case was that of a child, aged 5½ years, whose previous health had been good up to five weeks before admission to hospital. For five weeks she had suffered from severe pain in the head, mostly referred to the forehead, and from occasional vomiting. During the three days that she was under observation, besides this severe headache which caused the child to scream incessantly, sometimes for long periods, there was double optic neuritis, tache cérébrale, retracted abdomen, and irregular pulse. The diagnosis was tuberculous meningitis, and after the child had been three days in hospital, as it was believed that compression signs were imminent, it was decided to open the subarachnoid space by the occipital operation. A curved incision 2½ inches long was made over the left cerebellar fossa of the occipital bone with convexity upwards beginning below and behind the mastoid process, and ending at the external occipital crest. The trephine, of ¾ inch in diameter, was applied midway between the external occipital crest and the mastoid. The dura mater bulged, and there was no pulsation. After the dura mater and arachnoid were incised, thirty drops of serous slightly greenish fluid escaped, and the cerebellum came into relief. A silver probe with the terminal half-inch bent to a right angle was introduced between the cerebellum and the arachnoid, and directed inwards towards the falx cerebelli. When the falx was felt the probe was rotated, so that the end projected into the subarachnoid reservoir between the cerebellum and the medulla. Several drachms of serous fluid escaped. A drainage-tube was passed along the probe and left in position, and it was found that the liquid ran slowly along it. The dura mater was sutured. The disc of bone removed was cut into pieces and packed in mosaic in the wound, and a hole was left for the tube which pierced the skin. The pulse rose from 80 to 120 after the removal of the fluid. The temperature became normal, but afterwards varied between the normal and 103°. For some days there was a free escape of sero-sanguinolent fluid. The wound nearly healed; but afterwards it broke down, presenting gelatinous granulations. Ultimately, in about five weeks' time, there was sound recovery. During this interval the headache had vanished, the optic neuritis had gradually subsided, and the child had passed through the illness without any convulsions and without any mental failure. She subsequently passed through an attack of measles also, without any complication; and her brain condition remained satisfactory. Concerning this important case it is quite clear that, as the authors maintain, there was meningitis with some exudation collected at the base in the subarachnoid space; and it is highly probable that recovery was due to the surgical procedure adopted. But the evidence that the meningitis was tuberculous is by no means conclusive. It is explicitly stated that there was no sign of antecedent or subsequent pulmonary, abdominal, or cervical gland disease. Moreover the symptoms given, though suggestive of meningitis, hardly conform to the usual clinical picture of tuberculous meningitis—at all events, of the basic form. Severe head pain of five weeks' duration, if due to tuberculous meningitis, would usually have been followed or accompanied by several other manifestations, such as sighing, breathing, ptosis, strabismus, temporary facial palsy, and limited convulsions; but these were conspicuously absent. No examination of the fluid was made for tubercle bacilli; some of the granulations were removed from the sides of the wound after it had broken down, these were examined for tubercle, but with negative result.

Although the tuberculous nature of this case is debatable, its importance cannot be gainsaid. Previous writers who have urged the desirability of surgical interference in tuberculous meningitis, either by draining the lateral ventricles or the subarachnoid space, have for the most part based their arguments on the advantage of lessening intracranial pressure. Messrs. Ord and Waterhouse, drawing an analogy from the benefit which sometimes occurs from simple laparotomy for tuberculous peritonitis with effusion, urge that the removal of fluid in tuberculous meningitis may be advantageous in lessening the activity of growth of the tuberculous granulations. My own belief is, that in tuberculous meningitis the part played by the hydrocephalus has been exaggerated, and that the deleterious effect of the increased fluid pressure is not more important than the widespread cerebritis and its attendant softening which are brought about by the spread of the tuberculous invasion from the pia mater to the brain tissue.

Experience seems to show that the drainage of a moderate effusion in the lateral ventricles may be followed by excellent results if there be no widespread and severe damage to the brain tissue; and again the results of surgery have proved that, with proper methods of evacuation, the brain may recover from a local necrosis of moderate extent. But the widespread cerebritis and softening which, together with hydrocephalus and an extensive plugging of small vessels, sum up the conditions before us in unmistakable tuberculous meningitis, would appear too formidable a combination for any surgical procedure to remove.

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T. B.

SIMPLE MENINGITIS IN CHILDREN

(A) POSTERIOR-BASIC (OCCLUSIVE) MENINGITIS; ACQUIRED HYDROCEPHALUS.[1]

(B) VERTICAL AND CEREBRO-SPINAL (NON-OCCLUSIVE) MENINGITIS.

TUBERCULOUS meningitis has been carefully studied by many observers, and its symptoms and general course are well understood. But inflammation of the membranes of the brain and spinal cord occurring in infancy and childhood, in which the tuberculous process takes no part, has not attracted an equal amount of attention. We therefore propose in the following article to analyse the observations which we have made on this subject during the last twenty years at the Hospital for Sick Children and elsewhere, and shall endeavour—(i.) to discriminate between the different forms of the disease, (ii.) to elucidate, as far as possible, their pathology and treatment, and (iii.) to prove that hydrocephalus frequently results from chronic simple meningitis, and to explain how this comes about. The cases on which this article is based are 110 in number; they were all under the care of one or other of the authors, and many were seen by both. Fifty-nine were fatal, and in fifty a necropsy was obtained. We think that more advantage will be derived from a careful study of a sufficient number of cases recorded on a uniform plan by the same observers than could be obtained from comparison of a larger number of miscellaneous cases collected from medical records.

General summary of results.—In this article we consider solely the disease defined as non-tuberculous leptomeningitis, in which the morbid products are derived from the vessels of the pia mater, and tend to accumulate beneath the arachnoid in the subarachnoid space and in the ventricular cavities connected with it. We do not include cases in which the inflammation of the membranes is the result of diseased or fractured bone, or of syphilitic or other neoplasms. Neither do we include "Epidemic cerebro-spinal meningitis" (vol. ii. p. 659), although the more acute of our cases resemble that disease in many respects.

The cases may be arranged in two main classes—(A) the posterior-basic, and (B) the vertical.

A. *The posterior-basic class.*—To this group the majority of our cases belong. In them the inflammation begins at the posterior part of the base of the brain; they are therefore described as "posterior-basic." Leptomeningitis beginning at the anterior base is usually tuberculous. In most of the posterior-basic cases the primary site of inflammation is in the region where the brain and spinal cord unite, where the cerebellum overlaps the medulla oblongata. From this site the morbid process may extend downwards (in varying amount) around the spinal cord, upwards along the lining membrane of the ventricular cavities, and forwards along the base of the brain as far as the optic commissure and the tips of the temporo-sphenoidal lobes; but it usually spares the convexity, or affects it but slightly. Or the inflammation begins in the transverse fissure and the choroid plexuses, and is then usually less extensive. At an early stage the inflammatory exudation, though circumscribed, may be somewhat suppurative in character. If life be prolonged until the inflammatory process has subsided, a large part of the products of inflammation may be absorbed; but opacities of the meninges and adhesions are apt to remain. Opacity in the arachnoid of young children, except in the neighbourhood of the superior longitudinal sinus, is, we believe, a certain indication of former inflammation, not the result of any merely degenerative process. Adhesions may unite the cerebellum more or less firmly to the medulla, or may obliterate the iter (or even the fourth ventricle). The former result closes the three foramina by which the ventricular system of cavities communicates with the general subarachnoid space; the latter shuts off the third and lateral ventricles from the fourth. Both results are followed by an accumulation of cerebro-spinal fluid in the lateral ventricles, in other words by a *hydrocephalus.* In many instances the fluid which thus distends the ventricles is nearly transparent; in these the accumulation is almost wholly due to obstruction. In other cases it is evidently in part of inflammatory origin, being opaque and containing large flakes of inflammatory lymph, sometimes even pus; when this is so, it will often be found that the lining membrane of the ventricles is much thickened and shows distended vessels.

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[1] Congenital hydrocephalus will be dealt with later, by Drs. Shuttleworth and Fletcher Black, under the head of Idiocy and Imbecility.

In this form of meningitis the viscera are usually normal, or there is merely a little collapse found in the lungs.

Clinically, cases of this form are almost invariably signalised by one characteristic symptom—retraction of the head. This occurs at an early period of the illness, and continues until death, or, in the event of recovery, for several weeks.

"Convulsions" are often said to have occurred in the early stage of posterior-basic meningitis, but the descriptions given of these attacks lead to the conclusion that they often consist more of tonic than of clonic spasm. Definite clonic spasms of the extremities are less frequent in this disease than in vertical meningitis. On the other hand, tonic spasm is eminently characteristic of posterior-basic meningitis. As above stated, retraction of the head is almost always present, and this may be the only form of tonic spasm observed throughout the illness. But in many cases there is also rigidity of the limbs, sometimes considerable in amount, and in a few well-marked opisthotonos. Temporary exacerbations of the persistent tonic spasm, in which for a minute or two the head becomes more retracted, the back more arched, and the limbs more stiff, are not uncommon in the more severe cases; they remind the observer strongly of the phenomena of tetanus.

Vomiting is usually an early symptom of basic meningitis, often the first; and it may recur frequently throughout the illness.

The ocular symptoms are important. Persistent retraction of the eyelids, producing a fixed staring look, is sometimes well marked. In other cases clonic spasms of the eyelids are seen. Strabismus and nystagmus are fairly frequent; the former perhaps less common, the latter much more common, than in tuberculous meningitis. The pupils are often contracted at an early stage of the disease; at a late stage they are frequently dilated. Optic neuritis rarely occurs—a most remarkable fact in view of the long-continued basic inflammation, and in strong contrast with its frequency in tuberculous meningitis. Nevertheless the patients often become quite blind for a time. In patients who recover, the sight is usually restored.

Torpor may occur as an early symptom, and may then be transitory; but it may gradually increase and deepen into coma: in this case it is usually due to increasing intracranial tension caused by hydrocephalus. If recovery follows, the torpor slowly disappears, even in some cases in which hydrocephalus persists.

The fatal cases of this class last longer, as a rule, than cases of meningitis due to tubercle. Not a few recover more or less completely. Sometimes the recovery is quite complete: more commonly it is imperfect, and often attended with considerable enlargement of the head and obvious hydrocephalus. The child is backward in talking and walking; its mental development is frequently defective, often it is little better than an idiot. At a later period it may die suddenly, from little or no obvious cause.

B. *The vertical class.*—In a smaller number of cases the inflammation mainly affects the convexity of the brain. It is often most marked about the anterior part of the brain, and the region of the cerebro-spinal foramen may entirely escape. In the earliest stage there may be simply dryness and slight opacity of the pia arachnoid, with hyperæmia. At a later stage an œdematous condition may be present, with flakes of lymph along the vessels. But even within a few days there may be extensive inflammatory exudation, mainly suppurative in character, covering the whole of the convolutions of the convexity in one uniform sheet beneath the cerebral arachnoid. The base of the brain may be more or less implicated, and the spinal membranes involved. The ventricles are frequently normal; in some cases they share slightly in the inflammatory extension, and may contain a little purulent lymph; but, as a rule, they are not distended in this form of meningitis.

In vertical meningitis, inflammatory conditions of the viscera and serous membranes often coexist; especially pneumonia, empyema, and pericarditis. In cases with clinical evidence of one or more of these visceral inflammations a fatal vertical meningitis may suddenly supervene.

Cases of this class are often difficult of diagnosis, and the symptoms are sometimes remarkably latent. Retraction of the head is absent or very slight. Ocular symptoms are rare. Vomiting is less frequent than in the basic cases. Convulsions may occur, and may be violent and repeated and associated with high temperatures; but in some cases they are entirely absent. When present they are of the epileptiform type; the clonic spasms may be local at first, but they tend soon to become bilateral and general. We have never found in these cases the permanent tonic spasm of limbs or back, with temporary exacerbations, sometimes seen in posterior-basic meningitis.

The duration of vertical meningitis is usually much shorter than that of the cases in which the posterior base is mainly affected, shorter even than the three or four weeks which bring tuberculous meningitis to its inevitable end. It may be a fortnight, or not more than a week, or, as in three of the cases related below, as little as two days. It is not uncommon for death to occur in cases of this kind without any symptoms to lead to a diagnosis of meningitis. If the opinion has been formed that a case is one of vertical meningitis, it is sometimes impossible to decide whether the disease is or is not tuberculous until a necropsy is made. Only in very exceptional cases is it chronic.

We now proceed to give an account of the fifty autopsies. The cases are arranged in an order determined by the duration of the illness, beginning with those which were most rapidly fatal. This arrangement brings out clearly the differences in the condition found after death in the cases of shorter and longer duration respectively. It will be observed that the former are usually characterised by the presence of recent inflammatory lymph or pus; the more acute the case, the more purulent the exudation. In the protracted cases the lymph tends to become organised and to form adhesions, and hydrocephalus is often present. The shortest cases of all are those which are wholly or mainly vertical in distribution; in them the exudation is largely suppurative. The posterior-basic cases are rarely very short, frequently they last a long time; in these the exudation is more fibrinous, and in cases which have lasted for several weeks only adhesions and thickening and opacity of the membranes are found.

It will be of advantage to explain here that by the term "posterior subarachnoid space" we mean the large space (or "cistern," to use the term of Axel Key and Retzius) normally filled with cerebro-spinal fluid, bounded above by the convex line of attachment of the arachnoid to the pia on the cerebellum, and behind by the arachnoid as it passes from the cerebellum to the cord, which we term the "posterior arachnoid bridge." This space is continuous in front with the subarachnoid space of the brain, below with the subarachnoid space of the cord, and thus with the lymphatic sheaths of all the blood-vessels of the brain and cord, and also with the sheaths of all the outgoing nerves, both cerebral and spinal. It has also very important communications with the ventricular system of cavities by means of three foramina in the fourth ventricle—the cerebro-spinal foramen ("foramen of Majendie"), in the median line at the lowest point of the fourth ventricle, and two lateral foramina ("foramina of Luschka"), one in each lateral recess, close to the roots of the vagus nerve. It will be proved by the records which follow that when this "posterior subarachnoid space" is blocked with inflammatory lymph, or when adhesions resulting from previous inflammation close the above-named foramina, fluid collects in the ventricular cavities and a hydrocephalus results. A similar result may be caused by an obstruction at a higher or lower level, as will be explained hereafter. But when, as in the acute suppurative cases of vertical meningitis, this space is but little invaded, and the foramina are still patent, little or no excess of fluid may be found in the ventricles, and they are but slightly or not at all dilated. Such cases, indeed, are usually fatal before adhesions have had time to form.

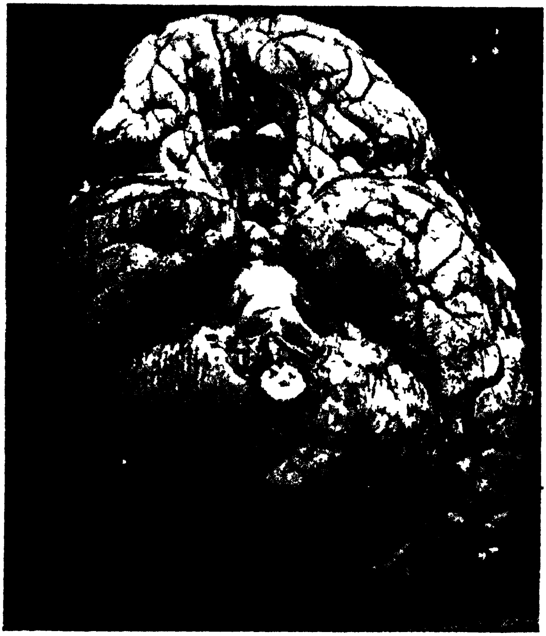

FIG. 27.—Photograph of the brain from which the coloured drawing was taken. The cerebro-spinal fluid obtained from the lateral ventricles during an operation, and also the fluid found in them after death, gave pure growths of the diplococcus described by Dr. Still in this paper.

We have added the date of each of the autopsies, and regret the imperfection of some of our older records. In every autopsy a careful search was made for tubercle, both in the cerebral membranes and in the viscera and glands. In only one of the cases was any tubercle found, and in this instance (P.M. 23) it was limited to the glands, thyroid, and spleen.

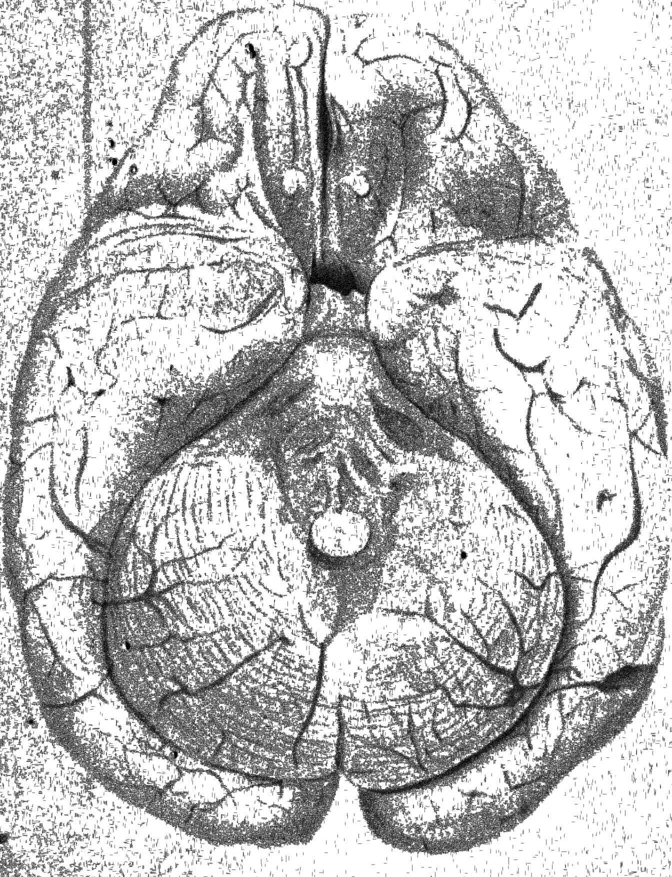

PLATE III

Base of the brain from a case of posterior-basic meningitis (age 5 months, duration 11 days). The exudation extends as far forwards as the optic commissure, and involves the tips of the temporo-sphenoidal lobes.

[*Face p.* 496.

POST-MORTEM RESULTS IN FIFTY CASES OF MENINGITIS[1]

We have once (in 1883, in a case of retraction of the head of a few days' duration) met with what may have been the earliest recognisable change of commencing vertical meningitis—a general cortical hyperæmia. The whole cortex was very red, especially in the posterior region, and a ramiform network of distended capillaries could be seen, but without any inflammatory effusion visible to the naked eye. We have not included this case in our analysis.

In a case observed in 1887 (age 4½ years; ill only forty-seven hours; vomiting, convulsions, dilated pupils, conjugate deviation of head and eyes to the left, rise of temperature to 107°, stiffness of neck, and slight retraction of head) we found greenish lymph over parts of both hemispheres adjoining the median line, some small ecchymoses on the frontal lobes, and a very little lymph on the superior vermiform process of the cerebellum. There was slight opacity of the arachnoid at the interpeduncular space. The region of the Sylvian fissures, the pons, medulla, and posterior base were normal. The brain-substance was soft. There was slight excess of fluid in the lateral ventricles, the other ventricles were normal. There was no lymph about the spinal cord, but some turbid fluid beneath the spinal arachnoid from the mid-dorsal region down to the cauda equina. There was slight collapse of the bases of the lungs, but no pneumonia. The spleen was slightly enlarged and soft. A few fatty patches in the liver. Other viscera normal. It is just possible that this case may have been one of epidemic cerebro-spinal meningitis, for some other cases of meningitis occurred about the same time in the same street.

In a second case (1896; age 10 months; bronchitis of uncertain duration, clinical evidence of broncho-pneumonia; vomiting twice the day before death; on day of death the fontanelle slightly bulged and markedly pulsating; no other cerebral symptoms) the pia arachnoid looked turbid, yellowish, and œdematous, and there was greenish-yellow puro-lymph along the vessels up to the vertex, especially about the right motor area. Brain-substance soft. Only slight excess of fluid in the ventricles. No lymph or pus on spinal cord, but its pia mater in lumbar region was slightly opaque. Both tympanic cavities contained muco-pus; otherwise normal. There was thick yellow lymph in the anterior mediastinum, especially over the external surface of the pericardium. About two drachms of sero-pus in the pericardial cavity, and flakes of yellow lymph about the origin of the great vessels and along the coronary arteries. Valves normal. Some evidence of pleurisy over the base of both lungs. The apex of the right lung was quite solid with lobular pneumonia, and there was a second small pneumonic area near the apex of the left lower lobe; also some areas of collapse. Some shreds of lymph on the spleen. Mediastinal and bronchial glands congested. Other viscera and glands normal.

In a third case (1894; age 1 year 4 months; ill thirteen days with cough; convulsions and vomiting on the sixth and again on the twelfth day; consolidation of the right apex; during the last two days of life retraction of head, stiffness of neck, frequent vomiting; discs normal) there was slight recent meningitis over the vertex of the brain; surface sticky, and slight adhesions in the longitudinal fissure. Some muco-pus in one tympanic cavity. Recent pleural adhesions over whole of right lung, and thick flaky lymph and a little pus over its lower lobe. The whole of the upper and lower lobes of this lung were completely consolidated: the middle lobe was collapsed. Left lung and pleura normal. Other viscera and glands normal.

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[1] These cases are admitted in full, and are placed at the beginning of the article, by the desire of the authors.—ED.

In a fourth case (1894; age 1 year 10 months: ill fourteen weeks with cough, dyspnœa, irregular pyrexia, dulness and bronchial breathing at apex of left lung; after ten weeks marked improvement; in twelfth week worse; five days before death vomiting and diarrhœa; on the day before death convulsions and rise of temperature to 103°, right optic disc blurred; temperature at death 105°) there was a considerable amount of turbid fluid and some lymph over the whole vertex and over the anterior portion of the frontal lobes; also lymph and purulent exudation over the upper surface of the cerebellum and posteriorly. Brain-substance soft. Ventricles normal, containing clear fluid, not distended The lateral sinuses as they joined the torcular were filled with purulent disintegrating blood-clot, and the same condition existed in the longitudinal sinus. The tympanic cavities were absolutely normal. No abnormal condition was found in the nose. There was a little meningitis in the lower dorsal region of the spinal cord, and much turbid fluid escaped. Pericardium abnormally adherent to sternum; it contained about half an ounce of pus, and the whole heart was covered with great pieces of flaky lymph, the trabeculæ of which enclosed pus. Heart not hypertrophied; valves normal. Both pleuræ universally adherent with firm adhesions of long standing; firmest on the left side. Much collapse in the left lung, and its upper portion was tough, but no part of it sank in water. The right lung appeared healthy. Bronchial glands somewhat enlarged. Other viscera and glands normal.

In a fifth case (1896; age 5 years 11 months: sudden onset of illness with rise of temperature to 104° nineteen days before death; cough and diarrhœa throughout; during the last three days of life headache, vomiting, and discharge of clear fluid from right ear, but no convulsions or retraction of head) the entire brain—cerebrum and cerebellum, vertex and base—was completely covered with greenish-yellow purulent exudation Ventricles appeared normal. Cord not examined. Ears not examined. Trachea congested. Bronchial tubes greatly congested and containing frothy red mucus. Lower lobe of both lungs covered with creamy lymph, and adherent to chest-wall and to diaphragm; the right lower lobe was intensely congested and consolidated, though it floated in water; the left showed only general bronchitis. There were yellow fibrinous clots in the right ventricle, and a very large one, firmly attached, filled the right auricle.

In a sixth case (1897; age 1 year 8 months; ill five weeks with cough, fever, and dyspnœa; five days before death convulsions, followed by weakness of right arm and leg, and occasional internal strabismus of right eye, temperature rising gradually to 105°) the entire cerebrum, vertex and base, was covered with yellowish-green puro-lymph, and similar exudation was found on the posterior arachnoid bridge and on the upper anterior part of the cerebellum But the cerebro-spinal foramen was not occluded, the ventricles were not distended, and their ependyma was normal. The cerebro-spinal fluid in them was not in excess, and was clear. Spinal cord covered with thick yellowish-green puro-lymph in nearly its whole extent. Both tympanic cavities contained viscid slightly turbid secretion, otherwise normal. Left pleura densely adherent throughout; posterior to the nipple line there was thick yellow puro-lymph over the middle portion of the lung posteriorly, covering part of the upper and part of the lower lobe. Lung tough, evidently compressed and collapsed, but not definitely pneumonic. Right pleura showed old adhesions; lung somewhat tough in parts. Pericardium externally abnormally adherent to left pleura and parietes. It contained about half an ounce of slightly turbid serum, and there were flakes of yellow lymph at the base about the great vessels, and some very recent adhesions. Slight loss of gloss over right auricle, ventricular surface normal. Other viscera normal, except that the sigmoid flexure was abnormally long. Glands normal.

Bacteriological examination by Dr. Still.—Pus from the pia of the vertex showed that the diplococcus pneumoniæ was present here in pure growths. Similar diplococci were found abundantly in the lymph from the pleura and in that from the pericardium. The mucus in the ears showed also similar diplococci, and a motile bacillus, probably the B. coli communis.

In a seventh case (1887; age 2 years 4 months; ill six days, head-retraction intense, two slight convulsions) there was greenish lymph on the anterior base, and much over the front of pons and medulla, and on the sides of the medulla, and some on the superior vermiform process of the cerebellum; some also on the convexity of the cerebrum, and much about the spinal cord (dorsal mainly). Ventricles slightly distended with blood-stained serum; dilated vessels on their walls. Cerebro-spinal foramen not obstructed. Slight collapse of the bases of the lungs. Slight fatty change in the liver. Other viscera normal.

In an eighth case (1895; age 4½ months; ill eight days; on second day vomiting; on third day a convulsion, fontanelle full, child apathetic and drowsy, pulse 220, respirations 30, temperature 98·6, rhonchi in both lungs: on fourth day vomiting and temperature 105°·8; on seventh day coma, some rigidity of limbs and convulsions; no retraction of head) there was thick smooth greenish-yellow lymph covering the anterior part of the vertex, very little over the posterior part. Similar lymph over anterior part of both temporo-sphenoidal lobes; some also over interpeduncular space, and a slight amount bridging over the Sylvian fissures. There was a small patch of similar lymph on the anterior part of the superior surface of the cerebellum, some on the under surface of the cerebellum, and the posterior arachnoid bridge was opaque and yellow with it. Convolutions slightly flattened. Not much fluid in the ventricles. No thrombosis of the sinuses. The spinal cord had a coating of similar lymph, especially in the lumbar region. The left tympanic cavity contained slightly turbid mucus; otherwise normal. The right contained a piece of semi-solid lymph very like that on the brain; the tympanic membrane was congested, but the ossicles were normal; no disease of petrous. Both lungs showed many small areas of mixed congestion and collapse; small pieces just sank in water. Other viscera and glands normal.

In a ninth case (1897; age 10 months; ill 16 days with cough; vomiting on 8th, 9th, and 10th days, not afterwards: no convulsions or retraction of head; fontanelle depressed during last two days of life) there was commencing meningitis (an œdematous appearance, with yellowish turbidity, and streaks of yellow lymph along the sulci) in the pia of the vertex, and on the anterior parts of the temporo-sphenoidal lobes. Posterior base, cerebellum, and ventricles normal. Yellowish turbidity on upper part of spinal cord on its posterior surface. The right tympanic cavity contained a considerable amount of thin pus; tympanic membrane slightly opaque; ossicles normal. The left contained muco-pus, otherwise normal. The left pleura contained about three ounces of thin pus; the right showed only a very small amount of lymph on its surface, no fluid. Upper lobe of right lung completely consolidated, and on section of a grayish-yellow colour. In the centre of the upper part of this lobe were two necrotic areas, with limiting membrane like a thin layer of inspissated lymph, and containing soft reddish-brown debris, but without any offensive odour. Left lung showed some patches of collapse and of slight broncho-pneumonia. Pericardium contained about half an ounce of thin sero-pus, with some flakes of lymph about the great vessels at the base; no adhesions; evidently very recent. Other viscera and glands normal.

Bacteriological examination by Dr. Still showed the presence of an apparently pure growth of capsulated diplococci—almost certainly diplococcus pneumoniæ—(1) in the lymph on the pia mater of the brain; (2) in the secretion and pus pressed out of the consolidated portion of the right lung; (3) in the lymph in the pericardium.

In a tenth case (1883; age 4 months; ill ten days, commencing the day after vaccination which ran a normal course, head held stiffly, vomiting, slight evidence of broncho-pneumonia, no convulsions) there was thick puro-lymph over most of the vertex, also at the posterior base and over the pons and medulla, as far forwards as the optic chiasma; some also on the under surface of the frontal lobes. Viscera normal except for slight pulmonary collapse.

In an eleventh case (1896; age 2 years 3 months; ill twelve days; ulcers on buttock and scalp, erysipelas, cellulitis, retraction of head, vomiting, and on day of death convulsions) there was yellowish-green lymph all over the vertex, especially posteriorly; a less amount all over the base, and a very slight amount on the upper anterior surface of the cerebellum; some also on the posterior arachnoid bridge. Ventricles contained a little slightly turbid fluid, but were not dilated. Ependyma normal. Similar yellow-green lymph along the spinal cord in the dorsal region. Tympanic cavities contained muco-pus, otherwise normal. In the lungs were small patches of collapse and one or two small areas of commencing broncho-pneumonia. Some flaky lymph on the spleen. Other viscera and glands normal; deep unhealthy-looking wounds (incisions) in left inguinal and gluteal regions.

In a twelfth case (1893, age 11 months; ill fourteen days, vomiting, diarrhœa, convulsions, slight head-retraction) we found a thin layer of greenish lymph beneath the arachnoid over the whole brain, both over the base and over the entire convexity. On section it was very thin, and passed from one convolution to another without dipping down into the sulci; it occupied the velum interpositum, but did not extend into the choroid plexuses. The cerebro-spinal foramen was open, about 4 mm. in diameter. There was lymph about the lateral foramina, and the brain was so soft that they could not be made out. Lateral ventricles not much dilated; they contained turbid fluid, and in the descending horns some puro-lymph. Six days before death the needle of an exploring syringe had been passed through the anterior fontanelle, first on the left side downwards to a depth of an inch and a half, afterwards on the right side downwards and slightly outwards to a depth of an inch and a quarter, the only result being a little bleeding from the first puncture. The track of the needle on the left side was detected by a hæmorrhagic fine line which very nearly, perhaps quite, reached the roof of the lateral ventricle. The track of the needle on the right side was less distinct, but near it extending outwards was a patch of hæmorrhagic pachymeningitis, about two inches in diameter. The longitudinal sinus was tightly blocked in its whole extent with partly decolorised clot, with extensions into the entering veins. The subarachnoid space of the cord contained yellowish lymph in nearly its whole length ; the cord was firm and on section normal. Right tympanic cavity contained muco-pus ; left normal. No carious bone. There were scattered patches of broncho-pneumonia throughout the left lung and at the base of the right. Liver and spleen congested. Two infarcts in the left kidney. Glands normal.

In a thirteenth case (1880 ; age at onset 7 months ; ill nineteen days, head-retraction, no convulsions, paralysis of the left facial, no otorrhœa) we found recent lymph at the posterior base, *blocking the posterior subarachnoid space.* It extended over the pons and medulla, involving the cranial nerves, and forwards as far as the optic chiasma and the Sylvian fissures. The *ventricles were distinctly dilated* and contained clear fluid Drum-membranes intact. Spinal cord not examined. Viscera normal.

In a fourteenth case (1885 ; age at onset 4 months ; ill twenty-one days) there was thick green puro-lymph over the convolutions of the vertex, especially over the frontal and temporo-sphenoidal regions on both sides, also over the anterior base, hiding the optic commissure and Sylvian fissures, and over the pons, and to a slight extent on the cerebellum. There was very little on the medulla, but the cerebro-spinal foramen was closed by very fine adhesions which broke when the brain was removed. The fourth ventricle was only slightly distended. But the *lateral ventricles were considerably distended with turbid fluid*, and *the iter was completely obliterated* by inflammatory adhesion ; *it could not be traced at all.* Spinal cord normal. Both tympanic cavities full of inflammatory lymph ; lining membrane of both granular and pink. Drum-membranes intact. Viscera normal except for two patches of consolidation in the left lung. It is noteworthy that in this case, although it was mainly of the "vertical" type, there was considerable distension of the lateral ventricles ; this was due partly to inflammatory extension, but mainly to the definite obstruction caused by the obliteration of the iter. Thus the case becomes a connecting link between the vertical and posterior-basic types.

In a fifteenth case (1894 ; age at onset 5 months ; ill twenty-four days, retraction of head, dulness at both bases, pus obtained by paracentesis from both ears) the whole of the anterior portion of the brain was covered with thick yellow puro-lymph, obscuring the convolutions ; this extended backwards as far as the posterior border of the Rolandic area. The posterior part of the vertex was free from lymph. Thick lymph covered also the whole of the anterior base of the brain, including the interpeduncular space ; behind this there was very little. There was some on the posterior surface of the cerebellum, but the space between the cerebellum and the medulla was quite free from it. Ventricles not distended. Puro-lymph extended all down the posterior aspect of the spinal cord. Mucous membrane of tympanic cavities swollen ; drum-membranes normal, save for incision in each through which pus had been removed from each ear a few hours before death. Nose examined and found normal. There was a considerable amount of patchy consolidation present at the base of both lungs. Other viscera and glands normal.

In a sixteenth case (1895 ; age at onset 1 year 9 months ; ill four weeks) there was no lymph or pus on the vertex, but the convolutions were flattened and the corpus callosum was bulged up by a large quantity of fluid in the ventricles. A large amount of thick green lymph covered the posterior base, extending from the cerebellum over the under surface of the medulla and pons ; it did not reach the Sylvian fissures, and there was only slight opacity of the membranes round the chiasma. *The posterior arachnoid cistern was distended*, and the lymph around the medulla obstructed the lateral openings, but the cerebro-spinal foramen was probably open. *Ventricles much distended* with turbid fluid, in which floated great flakes of lymph. Ependyma much injected. Brain soft. Thick green lymph surrounded the cord from the medulla to the cauda equina. Cord soft, but apparently normal. Tympanic cavities quite normal. Viscera and glands normal.

In a seventeenth case (1883 ; age at onset 9 weeks ; ill four weeks) we found recent lymph, becoming organised, at the posterior base, *obliterating the posterior subarachnoid space*, and slight opacity over the left temporo-sphenoidal tip, but none over the pons, anterior surface of medulla, cranial nerves, or remaining convolutions. The convolutions were extremely flattened, *and the ventricles all much distended* with fluid, which was for the most part clear but contained floating flakes of green lymph. Spinal cord not examined. Right drum-membrane opaque, mucous membrane of middle ear covered with thin granulations. Right Eustachian tube swollen and obstructed. Left drum-membrane clear ; mucous membrane normal. Left Eustachian tube normal. Viscera normal.

In an eighteenth case (1881 ; age at onset 6 months ; ill four weeks) while the skull-cap was being removed watery fluid poured out of an accidental wound of the brain ; it was collected, and amounted to six ounces. Convolutions flattened. *Lateral ventricles much dilated*, a very little yellow lymph found in the right. *Third ventricle and iter dilated. Fourth ventricle much dilated, and quite closed by lymph below.* Anterior part of brain, anterior base and nerves normal. On gently raising the cerebellum *in situ*, yellow lymph was seen beneath the arachnoid passing from the cerebellum to the cord ; there was a little lymph also on the anterior surface of the medulla, but none on the pons. A wedge-shaped piece of the occipital bone and the arches of the three uppermost cervical vertebræ were removed. The dura mater and the posterior surface of the arachnoid were normal, but *the posterior subarachnoid space below it was filled with yellow lymph* from the attachment of the arachnoid to the cerebellum down to the second cervical arch. At the level of the third cervical arch the cord and its membranes were normal. The rest of the cord was not examined. The left ear contained a little pus, the right none. Both tympanic membranes had been incised during life, and pus obtained from each. The lungs showed a little collapse. All the other viscera were normal.

In a nineteenth case (1884 ; age at onset 5½ months ; ill four and a half weeks, and treated for fourteen days with inunction of half a drachm of mercurial ointment twice daily) we found the "posterior arachnoid bridge" (the arachnoid covering the posterior subarachnoid space) turbid and spotty, and there were evident traces of inflammatory lymph over the front of the pons and medulla, but the posterior subarachnoid space was normal, there were no adhesions, the *cerebro-spinal foramen was widely open*, and the *ventricular cavities were not dilated.* The rest of the brain and the tympanic cavities were normal. Viscera normal except for some pulmonary collapse. In this case, therefore, the inflammatory process had been less acute, or else the products of inflammation had been absorbed ; and it is worth noting that the child had been treated with mercury for a fortnight. No obstruction having been caused, there was no hydrocephalus.

In a twentieth case (1887 ; age at onset 3¾ months ; ill five weeks) there was thick green puro-lymph over the base from the foramen magnum to the optic chiasma ; the basilar artery was completely embedded in it, and contained a firm thrombus. The posterior subarachnoid space was full of turbid fluid. The convolutions were flattened, and the *lateral ventricles much distended* with turbid fluid. *The iter was greatly dilated. The fourth ventricle was dilated, and so was the cerebro-spinal foramen ;* the lateral openings were wide enough to admit a penholder. The cause of this was found to be a *complete adhesion of the membranes to each other and the cord* in the cervical region ; the membranes could not be separated without tearing the cord. In the dorsal and lumbar regions of the cord there was a little thin pus in the subarachnoid space, mostly on the posterior surface. Both middle ears contained pus. The parietal regions of the skull showed extensive craniotabes. Ribs much beaded. Viscera normal.

In a twenty-first case (1894 ; age at onset 10 months ; ill five weeks) there were *adhesions about the upper part of the medulla*, and the posterior arachnoid cistern and all the ventricles were distended with clear fluid, *the lateral ventricles being much dilated.* The choroid plexuses were abnormally firm and hard, with a fibroid lump in each. Convolutions much flattened. Tympanic cavities contained muco-pus, otherwise normal. Spinal cord normal. Viscera and glands normal.

In a twenty-second case (1895 ; age at onset 1 year 8 months ; ill five weeks ; treated in hospital four weeks with a grain of iodide of potassium every three hours, and daily inunction of half a drachm of mercurial ointment) we found slight opaque thickening on the under surface of the left lobe of the cerebellum, but no adhesions. Pons and medulla normal. Anterior base showed slight opacity. Cerebro-spinal foramen patent and fourth ventricle not dilated. But the *lateral ventricles were considerably dilated with clear fluid*, and the convolutions flattened. Ependyma of ventricles not thickened. A small patch of rather thick yellow lymph was found on the anterior end of the superior vermiform process, proving that the meningitis had involved the vessels of the velum interpositum, and suggesting that though the iter was pervious *post mortem*, it *may have been compressed during life* by inflammatory effusion in its neighbourhood. Spinal theca somewhat distended with clear fluid. Distinct opacity and some thickening of arachnoid along the whole of the posterior surface of the cord. Tympanic cavities normal. Viscera normal.

In a twenty-third case (1892 ; age at onset 8 months ; ill five weeks) we found some lymph over the pons and in front of it, some also over the upper end of the left ascending frontal and first frontal convolutions, but none whatever at the posterior base, though there had been much head-retraction (with a tendency to turn the face to the right). The cerebro-spinal foramen was open, the cerebellum congested beneath the trephine hole, but no lymph over it. The ventricles were not dilated. The upper part of the spinal cord was normal ; the lower part was not examined. *The left ear was full of extremely viscid offensive puro-lymph*, extending into the antrum of the mastoid, in which the bone was slightly rough. The right tympanic cavity contained a smaller quantity of similar material. Ossicles present and normal on both sides. Drum-membranes entire; in the left one the scar of a healed incision was detected. There were no tubercles at the anterior base of the brain or in the Sylvian fissures, nor were any found in the lungs; but the bronchial glands were caseous, there was some tubercle in one cervical gland, and the spleen was full of miliary tubercles. There was also a small abscess in the left lobe of the thyroid, and a purulent sinus extending from it nearly to the epiglottis. The mesenteric glands were normal. The viscidity of the contents of the tympanic cavities explained why the paracentesis of the drum-membranes (nineteen days before death) was without effect. Two days before death a trephine-opening was made by Mr. Ballance over the left lobe of the cerebellum; only limpid fluid escaped, no lymph or pus. A few hours after the operation it was observed that the left eye was strongly drawn downwards, so that it was almost hidden beneath the lower lid; it was also affected with vertical nystagmus. The right eye was unaffected. Next day the two eyes were on the same level (about normal), but there was some vertical nystagmus of both. No lateral nystagmus.

In a twenty-fourth case (1897; age at onset 2 years 4 months; ill five weeks) there was some flattening of the convolutions, but the vertex was otherwise normal. The posterior arachnoid bridge was partially covered with lymph; this extended up to the attachment of the arachnoid to the cerebellum, ending there abruptly, except near the median line, where it crept up farther. The cerebro-spinal and lateral foramina were partially blocked. There was much thick whitish-yellow lymph on the under surface of the pons, extending almost to the chiasma; also a patch of it on the anterior inferior extremity of each temporo-sphenoidal lobe. Some lymph also on the anterior end of the superior vermiform process, and along the velum interpositum. Choroid plexuses too opaque in appearance, and veins somewhat distended. Ependyma slightly opaque. Brain-substance very soft. Iter appeared to be patent. Lateral ventricles considerably dilated with nearly clear fluid. Third and fourth ventricles not dilated. A thicker layer of similar lymph on the posterior surface of the spinal cord, and a little on its anterior surface. Both tympanic cavities contained some muco-pus, but were otherwise normal. Viscera normal.

In a twenty-fifth case (1895; age at onset 5 months; ill five weeks) we found adhesions of the cerebellum to the medulla, *blocking the posterior subarachnoid space.* There was a little thin lymph on the pons and medulla, at the anterior base, and extending up the Sylvian fissures; also a little on the cortex of the anterior part of the brain. The *ventricles were all distended* with puro-lymph, their ependyma very vascular and somewhat thickened. Brain rather soft. Puro-lymph was present also down the greater part of the posterior surface of the cord. Both middle ears contained muco-pus. Lining membrane of each tympanum vascular. Drum-membranes entire, showing scars of incisions. Viscera normal.

In a twenty-sixth case (1887; age at onset 11 weeks; ill six weeks, frequent fits of extensor spasm in all the limbs) we found *very firm adhesions of the membranes round the foramen magnum,* the dura mater being adherent to the posterior part of the under surface of the cerebellum, quite firm bands uniting them. *The posterior subarachnoid space was distended with green lymph,* its walls thickened. The cerebellum was hollowed out by the fluid pressure, and the pons and medulla squeezed into the closest possible apposition to the bone. There was very slight opacity in the interpeduncular space, none about the

Sylvian fissures. Convolutions very flattened; *ventricles much distended: iter apparently closed*, ependyma somewhat thickened. Brain soft. Spinal arachnoid greatly thickened, much lymph on posterior aspect of cord. The right tympanic cavity contained muco-pus, the left mucus. Viscera normal.

In a twenty-seventh case (1894; age at onset 6 months; ill six weeks; onset immediately followed a fall on the head) there were evidences of meningitis over the vertex and over the frontal lobes, also over the anterior part of the temporo-sphenoidal lobes, but none over the posterior part of the vertex, the cerebellum, or the pons, and none over the base except on the right temporo-sphenoidal lobe. No flattening of convolutions. No distension of ventricles. Brain substance normal; sinuses normal; spinal cord normal. Tympanic cavities contained muco-pus, otherwise normal. Some collapse in lungs. Other viscera normal. Death appeared to have been the result of persistent diarrhœa.

In a twenty-eighth case (1887; age at onset 1 year 2 weeks; ill six weeks) the recent lymph was most abundant at the anterior base and at the tips of the temporo-sphenoidal lobes, but some existed over the convolutions, also over the pons and medulla. Over the right hemisphere there were what appeared to be indications of traumatism, though no history of this had been obtained. There was some vascularity of the cranial surface of the dura mater in the right lateral region, extensive blood-staining of the lower surface of the dura mater over the right hemisphere, and beneath it, some lymph on the upper surface of the arachnoid, disguising the convolutions; this also was stained brown in places, probably from former hæmorrhage. There was also lymph on the upper surface of the cerebellum, and brown-stained lymph on its lower surface. The cerebro-spinal foramen was widely open. The ventricles were slightly distended, and contained some puro-lymph. There was thick lymph below the spinal arachnoid, most abundant in the dorsal and lumbar regions, almost entirely on the posterior surface. The right middle ear contained pus; drum-membrane destroyed, and external meatus plugged with swollen detached epithelium. The right petrous bone was vascular but not carious. The left middle ear contained pus, but the drum-membrane was not destroyed. The cranial bones were quite healthy and free from caries; no sign of fracture. We have no note of the condition of the viscera.

In a twenty-ninth case (1880; age at onset 7 months; ill seven weeks; treated for a month with a grain of hydrargyrum cum creta thrice a day and daily inunction of a drachm of unguentum hydrargyri) we found thin recent lymph at the posterior base, *closing the cerebro-spinal foramen*, but not filling the posterior subarachnoid space. There was thin lymph also on the pons, and a little on the under surface of the right frontal lobe and on the tip of the left temporo-sphenoidal lobe. *Ventricles distended with watery fluid.* Brain rather soft. Spinal cord not examined. Tympanic cavities both normal. Viscera normal.

In a thirtieth case (1879; age at onset 5 weeks; ill seven weeks) there was no recent lymph, and the posterior subarachnoid space was normal, but the *cerebro-spinal foramen was closed* by adhesion of the cerebellum to the medulla. *The fourth ventricle was much distended with clear fluid.* On opening it from above through the valve of Vieussens, the adhesion of the cerebellum to the medulla was found to extend all the way round except for one very small hole (probably torn during removal of the brain). When this was stopped, and water was poured in from above, the ventricle held water perfectly. There were also small traces of cicatrix over the pons and the tips of the temporo-sphenoidal lobes. The iter was pervious, *the lateral ventricles much distended with clear fluid*, the corpus callosum very thin, almost translucent. Not much flattening of convolutions, but fontanelle and sutures distended. Ears normal. Head only examined.

In a thirty-first case (1886 ; age at onset 7¾ months ; ill seven weeks) there were very slight adhesions between the lateral parts of the medulla and the cerebellum. There was no obstruction of the cerebro-spinal foramen, and the fourth ventricle was not distended ; but the choroid plexuses were much bound down, and there was thickening in the transverse fissure of the brain ; *the iter was quite impervious*, and the *lateral ventricles were greatly distended with clear fluid.* There was a little thin, partly organised lymph on the cerebellar peduncles, a little opacity of the arachnoid over the interpeduncular space and on the temporo-sphenoidal tips, and a few small patches of semi-organised lymph on the convexity. The brain-substance was softened, and the ependyma somewhat roughened. The spinal cord was normal (*though there had been marked opisthotonos*). Tympanic cavities contained muco-pus ; drum-membranes normal. Viscera normal.

In a thirty-second case (1883 ; age at onset 11 weeks ; ill seven weeks) *the cerebellum was closely bound to the medulla by thin adhesions ;* there was a little lymph on the under surface of the pons and medulla, and a very little on the under surface of the frontal lobes and on the tips of the temporo-sphenoidal lobes, but none along the Sylvian fissures or on the rest of the convolutions. *All the ventricles were distended with watery fluid*, with a very little pus in the third ventricle. Ependyma softened. Spinal cord not examined. Both tympanic membranes intact; the left was opaque, the right normal. There was pus in the left middle ear, and granulation-material on its mucous membrane ; some pus also in the antrum. Viscera normal, save for slight collapse of one lung and some emphysema of the other.

In a thirty-third case (1895 ; age at onset 4 months ; ill seven weeks ; rigid extension of right upper limb ; peculiar squealing cry) the posterior arachnoid bridge was found much thickened, resembling wash-leather, *and the cerebro-spinal and lateral foramina were firmly closed by strong adhesions.* The posterior subarachnoid space was empty. The subarachnoid spaces at the base of the brain, including the interpeduncular space, as far forwards as the optic commissure, were obliterated by organised lymph. Faint traces of opaque thickening on the tips of the temporo-sphenoidal lobes. The convolutions were flattened. *Lateral ventricles distended with greenish watery fluid*, with flakes of yellow lymph floating in it, and in the descending horns purulent fluid. Ependyma much thickened. Choroid plexuses shrunken into hard dark masses embedded in the thickened ependyma. Third ventricle contained fluid similar to that in the lateral ventricles. *Fourth ventricle entirely shut off from third by inflammatory adhesion at posterior end of iter ;* it was spherical in shape, and contained watery fluid, being *absolutely closed also below.* Thus the drainage system of the brain was converted into three cysts with differing contents, the lateral and third ventricles containing greenish watery fluid with fibrin and pus, the fourth ventricle containing clear fluid, and the posterior subarachnoid space being an empty cyst with thick walls. Brain-substance rather soft. Membranes of spinal cord adherent in front, and thickened and opaque posteriorly ; but there was no green lymph on the cord. The tympanic cavities both contained some pus, but the drum-membranes were intact, and the ossicles normal. Viscera normal.

In a thirty-fourth case (1882 ; age at onset 5½ months ; ill eight weeks) we found *the posterior subarachnoid space completely filled with flaky lymph.* Communication between the subarachnoid space of the brain and that of the cord was thus interrupted, but when this lymph was removed the cerebro-spinal foramen was found patent. *Ventricles moderately distended with slightly turbid watery fluid*, of which about 7 oz. escaped while the skull-cap was being removed. Ependyma of ventricles everywhere thickened, much injected, and flaked here and there with inflammatory lymph. This was specially marked in the descending horns of the lateral ventricles, which were thickened and matted together. Brain-substance soft. The arachnoid over the interpeduncular space was thickened and œdematous. The convolutions were flattened. The uppermost inch of the spinal cord appeared quite normal ; the rest was not examined. Tympanic cavities normal. Viscera not examined.

In a thirty-fifth case (1879 ; age at onset 4 months ; ill eight weeks) there was meningitis at the base from the optic commissure to the medulla ; much lymph, especially behind, where it was tougher and apparently older than elsewhere ; *it blocked the posterior subarachnoid space, and quite closed the cerebro-spinal foramen. Hydrocephalus was present.* The dilated ventricles contained some pus, as well as watery fluid, and their lining membrane was thickened. The right middle ear contained a little pus, the left was normal Spinal cord not examined. Viscera normal.

In a thirty-sixth case (1892 ; age at onset 11 months ; ill nine weeks) the convolutions were flattened and free from lymph. *Lateral ventricles, foramen of Monro, third ventricle and iter, all greatly distended with clear fluid.* The upper part of the fourth ventricle was distended with clear fluid, but *the lower part was obliterated by adhesions.* The lateral openings of the fourth ventricle were closed by fine membranes bulged up by fluid below. There was an opening about the position of the cerebro-spinal foramen, probably artificial, as two hours before death trephining had been done over the occipital bone, and an attempt made to drain the fourth ventricle ; the attempt failed owing to the presence of the adhesions in the fourth ventricle. Ependyma normal. Veins of Galen normal. The posterior subarachnoid space had been distended, and there was excess of subarachnoid fluid around the spinal cord, but no lymph. Four weeks before death both tympanic membranes had been incised, blood and pus escaping. One week before death the mastoid antrum had been cleared out on both sides. The petrous bones were soft and vascular ; on the left side there was a small opening into the skull along the petrosquamosal suture. Dura mater normal. Viscera normal.

In a thirty-seventh case (1881 ; age at onset 4 weeks ; ill ten weeks) *the cerebellum was found to be adherent to the medulla*, and *the ventricles all distended with a large quantity of watery fluid.* The cranium was extensively craniotabetic, and in several places was perforated, holes of considerable size existing, through which the cerebral membranes, distended by the fluid pressure within, projected as low soft swellings. These had been felt during life. The anterior fontanelle was very large, and the longitudinal suture open. The twin sister of this child was also under treatment for snuffling, great irritability, marked craniotabes, and slight enlargement of spleen. Great improvement in her condition was produced by daily inunctions of mercurial ointment.

In a thirty-eighth case (1878; age at onset 9½ months; ill ten weeks) we found a *delicate membrane across the cerebro-spinal foramen, completely closing it.* The cerebellum was also attached to the medulla on each side by fine adhesions, *closing also the lateral openings*, so that pressure on the medulla caused fluid in the fourth ventricle to bulge up the adhesions on either side without escaping. The arachnoid over the interpeduncular space was thickened and opaque. *All the ventricles were much distended with clear fluid.* The foramen of Monro admitted a forefinger; the iter admitted a lead-pencil; the fourth ventricle would have held a Barcelona nut. The closure of the exits from the fourth ventricle was tested by pouring water into the ventricle from above; it was found to hold water. Tympanic cavities both contained a small amount of semi-purulent fluid; drum-membranes intact. Head only examined.

In a thirty-ninth case (1887; age at onset 5 years; ill ten weeks; definite history of a fall on the occiput, followed the same day by pain in the head, and two days later by vomiting) there was some capillary injection on the outer surface of the dura mater, the vessels of the pia mater were injected, especially on the right side, and the puncta vasculosa more numerous than usual. There was some puriform fluid at the base, and lymph round the vessels in the left Sylvian fissure; some also over the pons and medulla, and streaks of it over the inferior surface of the cerebellum. The convolutions were flattened, and the ventricles much distended with clear fluid. Unfortunately, we have no note of the condition of the iter or of the cerebro-spinal foramen. The tympanic cavities and the spinal cord were not examined. The lower lobes of the lungs were congested, the upper emphysematous.

In a fortieth case (1877; age at onset 3 months; ill eleven weeks; extreme head-retraction; extreme opisthotonos; extensor spasm of limbs) the convolutions were flattened, and *the lateral ventricles greatly distended with clear fluid* (estimated at 10 oz.). The cerebellum was firmly adherent to the dura mater behind the foramen magnum by strong adhesions, which had to be severed with the knife before the cerebellum could be removed. *A firm sheet of thickened cicatricial membrane stretched across from cerebellum to medulla, leaving no aperture whatever.* Arachnoid over medulla thickened and œdematous. Cicatricial thickening at tips of both temporo-sphenoidal lobes, extensive on the left side. There was similar thickening about the edge of the velum interpositum, and a smooth membrane stretched across from the fornix to beyond the choroid plexus on each side, binding down these plexuses immovably. Many small distended vessels were seen passing into and under this membrane. When the membrane had been picked away the optic thalami could be made out, and two large distended veins (veins of Galen). The iter would have admitted a lead-pencil. Fourth ventricle distended. Upper part of spinal cord adherent to its dura mater. No further examination was permitted.

In a forty-first case (1896; age at onset 1 year; ill twelve weeks) the "posterior arachnoid bridge" was slightly adherent to the dura mater. It was distinctly opaque, especially above, where the arachnoid joins the pia on the cerebellum. The medulla was adherent to the cerebellum both inferiorly and laterally, *closing completely the cerebro-spinal and lateral foramina. Fourth ventricle considerably dilated; it was a closed cavity, the iter being obliterated above;* it contained clear fluid. *Third ventricle and foramen of Monro dilated. Lateral ventricles greatly distended with clear fluid.* Ependyma normal. Choroid plexuses rather hard and cord-like. Velum interpositum slightly opaque. Veins of

Galen normal. There was some opacity of the pia arachnoid in the interpeduncular space and about the optic chiasma, also in the Sylvian fissures, where there were some adhesions. A few small patches of opacity were found also on the frontal and temporo-sphenoidal lobes. Slight opacity of arachnoid on posterior aspect of spinal cord, and a little excess of subarachnoid fluid. Tympanic cavities quite normal. Viscera normal, except that the stomach contained some altered blood. Glands normal.

In a forty-second case (1887 ; age at onset 3 years 9 months ; ill twelve weeks ; origin possibly traumatic, a window having fallen on the back of his head two weeks before the onset) we found *the spinal membranes adherent to the cord in the uppermost cervical region.* Posterior arachnoid bridge showed spots of organised lymph. *Posterior subarachnoid space distended with clear fluid,* the pressure of which had slightly hollowed out the cerebellum on each side. *The lower part of this space was of conical form, the apex of the cone being formed by the adhesions of the membranes to the cervical cord.* Cerebro-spinal foramen dilated. Slight cicatricial opacity over pons and medulla, with cystic bulging at the position of the closed lateral foramina. Anterior base normal. Hæmorrhagic thin false membrane on arachnoid surface of dura mater over both hemispheres, and a few small hæmorrhagic spots over petrous portion of both temporal bones. Convolutions slightly flattened. *Ventricles all moderately distended with clear fluid.* Iter admitted a crow-quill. Brain-substance fairly firm. Upper part of spinal cord, below adhesion of arachnoid, was normal ; lower part not examined. Left tympanic cavity normal ; right lined with fine granulations. Drum-membranes both intact. Viscera normal.

In a forty-third case (1884 ; age at onset 3 years ; ill twelve weeks) the openings from the fourth ventricle were *closed by delicate but firm adhesions of the cerebellum to the medulla.* Some partly organised inflammatory exudation was found on the pons, and as far forwards as the optic chiasma, and the optic nerves were slightly bound down. There was none on the convolutions, but these were flattened. The ventricles contained perfectly clear fluid, *and were much distended.* Velum interpositum slightly bound down. *Iter dilated. Fourth ventricle dilated,* with distended membranes over the lateral horns, forming a pouch on either side close to the vagus nerve. These pouches represented the closed lateral openings ; they could be made more prominent by compressing slightly the fluid-distended ventricle. Spinal cord not examined. The right tympanic cavity contained some viscid fluid ; its lining membrane was thickened and pinkish ; drum-membrane opaque. Left tympanic cavity quite normal. Viscera normal, except that there was a little collapse at the bases of the lungs.

In a forty-fourth case (1896 ; age at onset 1 year 4 months ; ill thirteen weeks ; extreme head-retraction ; opisthotonos ; extensor spasm of limbs) we found the posterior arachnoid bridge adherent to the dura mater over it, but when separated it was thin and nearly translucent. The cerebro-spinal foramen was open, though it was smaller than usual ; the lateral foramina appeared to be closed. The fourth ventricle was much dilated. From it and the subarachnoid space of the cord 2 oz. of clear fluid were removed by a hypodermic syringe passed below the second lumbar vertebra at the commencement of the autopsy. When this ceased to flow, a fresh supply of clear fluid was easily obtained by puncture through the fontanelle, and it was found that *each lateral ventricle was a separate cavity distended with clear fluid, the foramen of Monro being quite obliterated on each side. The iter was also obliterated, and the third* *ventricle (thus closed both above and below) was distended with clear fluid.* So that in this case the hydrostatic system of the brain and cord was *severed into four sections*—the right lateral ventricle, the left lateral ventricle, the third ventricle, and the fourth ventricle and the subarachnoid space of the cord—all distended with clear fluid. Ependyma very slightly opaque. Slight opacity of the pia arachnoid about the chiasma and interpeduncular space and Sylvian fissures, and more markedly on the anterior end of the superior vermiform process of the cerebellum, but none on the temporo-sphenoidal lobes. Right ear normal; left ear contained a little semi-purulent fluid. Tympanic membranes normal. Spinal cord normal. Viscera normal.

In a forty-fifth case (1892; age at onset 9 months; ill fourteen weeks) the *cerebellum was completely adherent to the medulla.* The adhesions were firm and vascular, and formed a complete barrier to the passage of fluid from the ventricles to the subarachnoid space. *Fluid injected through the iter with a fine glass tube under a pressure of 2 feet of water did not pass at all through this barrier. Lateral ventricles greatly dilated*, and contained more than 10 oz. of clear fluid. Ependyma slightly granular. Foramen of Monro, third ventricle, iter, and fourth ventricle *all much dilated.* A small hernia of the cerebellum protruded through a trephine opening in the right occipital bone. This opening was made nine days before death; through it about 7 oz. of cerebro-spinal fluid had been removed from the base of the brain. Dura mater over the posterior subarachnoid space normal; arachnoid somewhat thickened and opaque. The subarachnoid space of the cord contained some excess of clear fluid. Tympanic cavities normal. Viscera normal, except for the presence of a Meckel's diverticulum.

In a forty-sixth case (1897; age at onset 3 months; ill fifteen weeks) the cerebellum was entirely adherent to the medulla inferiorly, so that the *cerebro-spinal and lateral foramina were closed by fibrous adhesions.* No recent lymph. The posterior arachnoid bridge was much thickened, opaque, and adherent to the dura mater. *In the mid-cervical region of the cord the dura, arachnoid, and pia were all matted together and firmly united to the cord.* The convolutions were much flattened, and the *lateral ventricles greatly dilated*, containing about 14 oz. of clear fluid. The foramen of Monro, third ventricle, iter, and fourth ventricle were *all greatly dilated.* Ependyma, choroid plexuses, and veins of Galen normal. The whole of the anterior surface of the pons was adherent to the bone. The arachnoid over the interpeduncular space was opaque and thickened, and distended by clear fluid below. There were adhesions over the surface of the Sylvian fissures, but none in their deeper parts. There was much opacity and thickening on the inferior surface of both temporo-sphenoidal lobes, especially at their anterior extremities. There were also patches of white thickening along the sulci almost up to the vertex, especially in the anterior part of the brain. Below the adhesions in the mid-cervical region the spinal membranes were almost normal. Tympanic membranes and ossicles normal; muco-pus in both tympanic cavities. The lower lobe of each lung contained areas of collapse, intermixed with areas which on section were maroon-red, with slightly granular surface, solid but friable, sinking in water. Anterior lower margin of left lower lobe emphysematous. Other viscera and glands normal.

In a forty-seventh case (1878; age at onset 8 weeks; apparently recovered after an illness of thirteen weeks, and gained flesh, though with occasional vomiting and some divergence of the eyes on going to sleep, and appeared to be going on well, but three months after apparent recovery was carried off in twenty-four hours by a severe attack of vomiting) we found the convolutions flattened and the *ventricles much distended with clear fluid. The cerebro-spinal foramen was quite closed by cicatricial membrane* uniting the superior part of the medulla to the cerebellum. This membrane showed two or three small areas of definite white cicatrix, also some small hæmorrhages near them. There were traces of cicatrix also on the tip of the right temporo-sphenoidal lobe (none on left). The tympanic cavities contained some turbid mucus, and one of them showed a little bare bone and some pale-red granulations. Viscera normal. No tubercle. Spinal cord not examined.

In a forty-eighth case (1879; age at onset 9 weeks; apparently recovered after an illness of ten weeks, but died unexpectedly seven months later after two days of slight catarrh, having in the interval appeared to be quite well) we found the *lateral ventricles equally and considerably dilated with clear fluid.* The corpora striata were finely granular on the surface, as if dusted with sand. A large thin translucent cyst was attached to, and apparently arose from, the left choroid plexus, occupying the descending and posterior cornua; it contained clear fluid. The right choroid plexus contained a few small imperfect cysts. The foramen of Monro admitted the end of a forefinger The iter could be made out, but was impermeable at its lower end. *The fourth ventricle was entirely obliterated*, the roof and floor being firmly adherent in their whole extent. The adhesions were very tough indeed, and separation could not be effected without tearing the nervous substance. There was no recent lymph. The cerebro-spinal foramen was open, and there was an entire absence of any morbid change in the membranes in its neighbourhood and over the pons, but there were a few small brownish spots in the pia mater over the anterior lobes of the brain, and over the tip of each temporo-sphenoidal lobe. Cervical cord and membranes normal; rest of cord not examined. The right tympanic cavity was lined with red thickened mucous membrane, which could be easily stripped from the bone; it contained muco-pus. The drum-membrane and the ossicles had disappeared. The right petrous bone was evidently inflamed, it was too vascular, and softer than normal. The left petrous was less vascular than the right, and the ossicles were present, but the drum-membrane was perforated, and the tympanic cavity contained muco-pus, and its lining membrane was like that on the other side. The dura mater over both petrous bones was normal, and there was no vascularity or lymph about the seventh nerves. Viscera and glands normal.

In the forty-ninth case (1881; age at onset 6 weeks; ill 11½ months) the illness began with a fit at six weeks old, followed by nystagmus, divergence of eyes, some retraction of head, and a month later by constant spasmodic movement of many muscles, which lasted four days. After this there was progressive increase in the size of the head, attended with persistent pain. At eleven months old the circumference of the head of this child, though a female and wasted, exceeded by nearly 3 inches that of a healthy male child of the same age; it amounted to 21 inches. The measurement over the vertex from one auditory meatus to the other was 14¾ inches, or 2 inches more than the average. The head was still larger when she died two months later. We found at the autopsy that the convolutions were entirely flattened out. No fluid in the subdural space. *Ventricles enormously distended* with clear fluid (estimated at two pints). The foramen of Monro was of the diameter of a half-crown. Third ventricle dilated. Iter admitted a lead-pencil. Fourth ventricle enormously dilated (size of a hen's egg). *Entire adhesion of the cerebellum to the sides and back of the lower part of the medulla, the subarachnoid space being here entirely obliterated by firm tissue.* The adhesion of the membranes continued down to the level of the third cervical vertebra. Below this the arachnoid could be separated from the pia mater. Viscera and glands normal. It might possibly be thought that this was a case of congenital hydrocephalus; but at three months of age, six weeks after the first symptoms, the head was still of normal size—the circumference being then 15½ inches, and the measurement over the vertex from meatus to meatus 10½ inches.

The fiftieth case (1891; age at onset 2 years) we have left to the last, because of the exceptional condition found at the autopsy. A boy, aged two years, after a fortnight's catarrh, became fretful, and vomited. The vomiting was repeated several times on the two subsequent days. On the second and third days he seemed only partly conscious, lay still, made no sound. On the fourth day he "screamed every five minutes for an hour, and his head became strained back." On several occasions it was observed that he "rolled his eyes, clenched his hands, and twitched both arms and legs." The head-retraction continued until death (ten weeks), and became very marked. After a time there was also marked opisthotonos, so that the occiput was brought to within 5 inches from the buttocks; this, however, diminished, and before death had almost disappeared, though the head-retraction remained. There was rigidity of all the limbs, at first slight and flexor, later and till death marked and extensor. The abdominal muscles were also rigid. For a few days the right hand was in the tetany position, but this soon passed off, and all the fingers of both hands were then flexed at every joint; during the last week the "claw-hand" was present, markedly on the right side, less on the left. The heels were rigidly drawn up, so that the dorsum of the foot on each side was on a line with the front of the leg; the toes contracted and the soles arched. There was at times slight horizontal nystagmus in both eyes, and occasionally convergent strabismus. The pupils varied in size, but remained equal; at first each measured about 5 mm., and reacted readily to light. Twelve days later they were less dilated, but did not react to light (no atropine for ten days). Next day they varied continually in size, but did not react to light. On the following day they were contracted, but still did not react to light. The optic discs were pale, the upper and lower margins a little indistinct, but the temporal edges defined. Both tympanic membranes were incised by Mr. Ballance nineteen days after the onset; only a little blood escaped, no pus, and the symptoms were not relieved. During the illness the child wasted greatly; on admission he was plump, but before death he became extremely emaciated, though he took (part of the time by nasal feeding) a fair quantity of nourishment, and had no vomiting.

The necropsy was made by Dr. Voelcker and Dr. Lees. The spinal cord, medulla, cerebellum, and the posterior part of the cerebrum were exposed *in situ* by the removal of the posterior wall of the vertebral canal and the back of the skull. The theca vertebralis was found distended in its whole length. On slitting it up, a small quantity of clear fluid escaped from the subdural space. The arachnoid was then seen to be distended with clear fluid, which filled the subarachnoid space from the "cistern" between the cerebellum and the medulla to the lower end of the cord. Its upper limit was marked by an opaque-white curved line in the pia arachnoid over the under surface of the cerebellum. The fluid was as transparent as water, without the slightest turbidity, so that the roots of the nerves (for example, the spinal accessory) could be seen with absolute distinctness through the perfectly transparent arachnoid. On detaching the cord below, some of the fluid escaped, and the distended arachnoid partly collapsed ; the brain and cord were then removed together. There was slight milky opacity over the upper end of the fissure of Rolando on each side. Surface of brain pale and convolutions flattened. No tubercles. Cerebro-spinal foramen patent and large (would have admitted a lead-pencil). Lateral foramen on left side patent ; that on right side appeared to be closed, and cerebellum seemed to be more adherent than normally to the medulla. Lateral ventricles much distended and contained clear fluid. Veins of Galen and their tributaries were pervious ; no thrombosis. Foramen of Monro enlarged, and third ventricle dilated. Iter patent but not dilated. *No definite obstruction found anywhere.* Both tympanic cavities contained mucus ; drum-membranes perfect (incision fifty days previously). Viscera and glands normal. No tubercle anywhere.

ANALYSIS OF SYMPTOMS.—**Sex.**—Of the 110 cases on which this paper is based, 58 were males, 52 females. The sexes are therefore equally liable.

Age at onset—

Under three months of age . .	11 cases.
Over three and under six months .	39 „
Over six and under nine months . .	19 „
Over nine and under twelve months .	15 „
During the first year	84 cases.
„ second year . .	13 „
„ third year . . .	6 „
„ fourth year . . .	2 „
„ fifth year	1 „
„ sixth year . . .	4 „
	110

Average age at onset of the 110 cases = 11 months.
„ „ „ 84 cases in first year = 5 months.
„ „ „ 26 cases older than one year = 2 years 6 months.

Eighty-four (76 per cent) of the cases were in the first year of life ; in sixty-nine (63 per cent) the illness began during the first nine months ; in fifty (45 per cent) within the first six months.

The average age at onset of the 14 most definitely vertical and suppurative cases was 20 months. This higher average was caused by the inclusion of two cases aged 4½ years and 5 years 11 months respectively. Excluding these, the average age at onset would be 13 months, very little higher than the average (11 months) of all the cases. But it may be noted that 50 per cent of the 14 were older than one year, while only 24 per cent of all the cases were above this age.

We may add that we have three times met with posterior-basic meningitis in older children, aged 6 years, 8 years, and 11 years respectively.

Month of onset.—In 85 cases (none of them of traumatic origin) we found the following numbers:—

January	14	July	3
February	11	August	2
March	9	September	5
April	10	October	3
May	10	November	6
June	8	December	4
			85

Sixty-two cases occurred in the first half of the year, only 23 in the second half—a ratio of nearly 3 to 1. This is a striking fact, and is perhaps due to the greater cold and variability of weather in our later winter and spring.

Previous health.—The majority of our patients had been in good health until shortly before the onset of the disease; but some were weakly, and others rickety. In about half of those of whose previous health we have notes (37 out of 76) the onset of the illness was immediately preceded by obvious signs of catarrh, either nasal, bronchial, or intestinal; and in six others the symptoms of meningitis set in soon after an infectious disease likely to have produced bronchial catarrh (measles, 4; German measles, 1; whooping-cough, 1). In one case varicella preceded the onset. We have also seen a few cases in older children in which symptoms of meningitis were associated with influenza. It is clear, therefore, that the occurrence of catarrh is a point of great importance in the etiology of the disease.

Inherited syphilis, on the other hand, seems to us to have very little share in the production of meningitis, apart from other lesions of the brain or its vessels, in young children. In only three of our cases was there strong reason to suspect that the child was syphilitic, and in a few more there was slight, but quite inadequate, evidence of this condition. In a paper by Dr. Gee and one of us (Barlow) on "The Cervical Opisthotonos of Infants," published in *St. Bartholomew's Hospital Reports* for 1878, there are 2 cases out 25 in which the existence of inherited syphilis was undoubted, but no necropsy was made. It must be allowed, however, that syphilis is a possible cause of meningitis. In an article by Dr. Barlow and Dr. Bury (1) are recorded autopsies on syphilitic children in which meningitis was found. In some of these instances it was localised in the neighbourhood of syphilitic lesions of bone and dura mater; in others it was generalised, and accompanied syphilitic endarteritis, gummata, or sclerosis. We have recently seen other similar cases. Their course is usually chronic and irregular, and rarely conforms to the types which we have described. But in one autopsy, on an infant aged 7 weeks, we found a softening gumma near the right optic thalamus, and also recent lymph over the pons and medulla which had closed the cerebro-spinal foramen; the ventricles were distended with fluid containing flakes of lymph; the brain was very soft. Head-retraction and convulsions had occurred during life. This case might have been included in our analysis but for the existence of a definite cerebral tumour, which excludes the case in accordance with our preliminary definition. Such cases are rare, and the greater number of instances of meningitis in syphilitic children are probably examples of the presence of two independent diseases in the same patient. Coincidences must occasionally be met with; the inheritance of syphilis affords no protection from catarrh and otitis.

Previous otorrhœa.—This was noted as absent in 57 cases. In one case otorrhœa occurred four weeks before the onset, but ceased two weeks before. In three cases otorrhœa was said to have been present from a very early age; in one of these, however, the onset followed a fall, and was probably traumatic and independent of the aural condition; and in another the tympanic membranes were intact at the autopsy.

Influence of dentition.—We could not prove any certain influence of dentition in the causation of this disease. In only seven cases have we notes of teeth being cut during the illness. Rarely did we find that the child had been observed to put its hand into its mouth.

It must be noted, however, that the number of cases occurring in the second three months of life (thirty-nine) is more than three times as large as the number (eleven) occurring during the first three months. Possibly this may indicate some influence of dentition, but we are more inclined to attribute it to greater exposure to cold.

Injury to the head as a cause.—This is a possible factor in the etiology of meningitis, even apart from fracture of bone; and careful inquiry sometimes discovers a clear history of the occurrence of a fall or a blow on the head shortly before the onset of the disease. Some of these occurrences are probably mere coincidences, but in a few cases the sequence seems to be one of cause and effect. For instance, one child "had a fall on its forehead. Was irritable after this. On the third day after the fall it screamed, and on the fifth day its head was retracted." A second, "fell from a girl's arms on the pavement on May 22nd. The fall was seen by the mother. The child was hushed to sleep, but did not move its head properly after this. Had been quite well before. Vomited the same day (after a powder). Retraction of the head marked since May 25th." A third child, 2½ years old, "fell on the back of her head on December 26th; did not seem to hurt herself; played afterwards. On the 27th she seemed to be ill. On the 28th a convulsion occurred, lasting three-quarters of an hour (hands and feet clenched), directly after which retraction of the head came on and lasted two weeks. Vomiting also soon after the fit." A fourth child was seen by its mother to fall on its head from a couch (the distance estimated at 15 inches). "An hour afterwards it vomited; it had not vomited before. Next day the vomiting recurred five or six times, and again on the third day. Head-retraction began on the third day." In a fifth case a fall on the head occurred "just before the onset of the disease, and a bruise was found on the vertex. The child seemed ill afterwards and vomited." A sixth child, aged 5 years, "was frightened by a fowl flying at her, and fell on her occiput. On the same day she complained much of pain in her head, but did not vomit. The pain continued. On the third day after the fall she vomited three times. Head-retraction commenced a few days later."

With regard to blows on the head one case seemed clear. "A broom-handle fell on the child's head at six o'clock one evening. He cried for ten minutes, and a red mark came out on his forehead. Vomited four times during the night. At eleven o'clock next morning a lump was found over his fontanelle, and he held his head back. The eyes have twitched ever since." In another case a "window came down on the back of the child's head" two weeks before the onset; but though no symptoms were said to have been noticed for a fortnight, yet the autopsy (P.M. 42) made it probable that the injury was really the cause of the fatal meningitis.

The first sign of the disease.—We have careful notes of this in 102 cases, and find on analysis :—

First sign	vomiting	33	cases.
,, ,,	convulsion	29	,,
,, ,,	head-retraction	24	,,
,, ,,	screaming or irritability	9	,,
,, ,,	sleepiness or languor	7	,,
		102	cases.

Whichever of these were the first sign, two or three of the others often followed on the same day, or a day or two later. At the same time it must be remembered that the history has to be obtained from the mothers, and may not always be quite accurate; also that the early stiffness of the neck is easily overlooked until the head becomes decidedly retracted. Still the figures seem to warrant the conclusion that in about one-third of the cases vomiting is the first indication, in one-third convulsion, and in one-third retraction of the head.

Early bulging of the fontanelle.—This was noticed in nineteen cases, sometimes by the mother only, at other times by ourselves also. In one child it was "prominent on the fourth day"; in another it was tense on the sixth day, and remained so till death four days later. In a third it had "been noticed by the mother since the fit at onset, and it remained distended when the child was seen three weeks later." In a fourth it was "tense and convex when first seen, thirteen days after fall on forehead and seven days after onset of head-retraction." In the case certainly due to a blow, "a lump" over the fontanelle was noticed seventeen hours after the injury. In a sixth case there had been "a lump" over the fontanelle for about two days after the child was first taken ill; it lasted a few days. In a seventh case there had been "a hard lump" in the same situation when the head was first drawn back; after three days it softened and went down. In an eighth there was "a hard lump" over the fontanelle "during the first part of the illness." In a ninth the fontanelle was a little bulging when first seen fourteen days after onset; the mother said it had been so for a week. In a tenth it was noticed by the mother at the onset, and gradually increased until the child was seen a week later, when the fontanelle was found to be full and the veins of the scalp large. In an eleventh it had been observed by the mother on the second day; it gradually lessened. It is clear, therefore, that undue fulness of the fontanelle in the first few days of the disease is a fairly common symptom; no doubt it indicates a considerable hyperæmia of the brain and its membranes, an "active congestion," and an increased outflow of fluid from the vessels.

Head-retraction.—This symptom, if early, marked, and persistent, is the most distinctive indication of a posterior-basic meningitis. It may occur in the later stages of tuberculous meningitis, but is rarely of intensity equal to what is common in simple meningitis affecting the posterior base of the brain. In vertical meningitis there may be no definite retraction of the head, though there is often some stiffness of the cervical muscles, flexor as well as extensor; so that the head does not tend to fall back when the body is raised from the bed. Our notes give the following information with regard to the date of the first appearance of head-retraction, its intensity, and its duration:—

Appearance on first day of illness .	34 cases.
,, second ,,	5 ,,
,, third ,,	7 ,,
,, fourth ,,	5 ,,
,, fifth ,,	2 ,,
,, sixth ,, . . .	1 ,,
,, seventh, or "a week after onset" .	5 ,,
"within the first week" . .	9 ,,
	68 ,,
Appearance during second week of illness .	3 ,,
,, ,, third ,, .	5 ,,
,, ,, fourth ,, .	1 ,,
	77

Thus in 34 cases out of 77, or nearly one-half, the retraction of the head began on the first day of the illness; and in 68, or ten-elevenths, before the end of the first week.

The intensity of the head-retraction is described as "very marked" in 13 cases, as "marked" in 23, as "moderate" in 17, and as "slight" in 8. In 45 its existence is noted, but not its amount; in all of these it was probably moderate or slight. It may be concluded, therefore, that in about half of the cases it is sufficiently great to excite immediate notice.

As to the duration of head-retraction, we find that it lasted—

	In fatal cases.		In cases of recovery.	
Not over 1 week	7		0	
From 1 to 2 weeks	3		6	
" 2 " 3 "	2		8	
" 3 " 4 "	4		6	
" 4 " 5 "	11		6	
" 5 " 6 "	5		4	
" 6 " 7 "	5		3	
" 7 " 8 "	3		1	
" 8 " 9 "	2		0	
" 9 " 10 "	1		3	
Over 10 "	1		10	
	44	+	47	= 91 cases.

The figures for the cases known to have been fatal are accurate; those for the cases of recovery are no doubt too low, for in some cases the patient was seen on only one occasion, and probably in many instances attendance ceased before absolute recovery, even as regards this single symptom. And some of these may have been fatal later.

The figures indicate that in one-fourth of the fatal cases the head-retraction lasts less than two weeks, but in one-half it lasts from four to seven weeks. On the other hand, in one-half of the cases which ended in recovery it lasted from one to five weeks, and in one-fourth over ten weeks.

The retracted position of the head is due to the tonic contraction of the posterior cervical muscles, probably of the complexus and other deep muscles rather than of the trapezius, on each side. Tense muscle can usually be felt distinctly in this position. An attempt on the part of the physician to bring the head forward is almost always painful to the child, and often causes a cry, especially in the earlier stages of the disease. Even ordinary handling of the child, as in washing it, causes much distress; only when left quite undisturbed does it seem free from pain. As the retraction of the head begins to pass off, this tenderness disappears; the head may then be brought forwards gently without causing any sign of pain. At a later stage still the head seems to drop back rather than to be drawn back, its position being now due apparently to weakness of the cervical muscles, and no longer to any spasm. Often it is difficult to say precisely when the one condition passes into the other.

Ocular symptoms.—1. *Strabismus.*—This is a frequent symptom. We have notes of its presence in 36 cases (convergent 17, divergent 11, direction not stated 8), and it was reported to have been present, but was not observed by us, in 5 other cases, making 41 in all. It is much less commonly due to definite paralysis of ocular muscles than is the case in tuberculous meningitis. Other abnormal positions of the eyes were noticed occasionally. In 1 case both eyes were turned upwards; in 2 cases both were turned downwards; in 3 there was conjoint deviation to the left; in 1 to the right; in 2 there were independent movements of the eyes. In several cases there was marked retraction of both upper eyelids, giving the eyes a staring appearance, such as is seen occasionally in exophthalmic goitre ("Stellwag's symptom").

2. *Nystagmus.*—This phenomenon is more common in posterior-basic meningitis than in tuberculous meningitis. It consists of oscillations of the eyeballs, usually conjoint, in rare cases affecting one eye only or almost so, spontaneous and casual and not merely during forced voluntary movement in one direction, as in insular sclerosis. In a few cases we have observed simultaneous contractions of the orbicularis palpebrarum. We noted some variety of nystagmus affecting both eyes conjointly in 20 cases (lateral in 6; vertical in 4; upwards and to the right in 1; rotatory in 1; direction not noted in 8). We observed nystagmus of the right eye alone in 1 case, and nystagmus of the right eye mainly with much slighter conjoint affection of the left in 1 case. In 4 cases in which we did not detect any nystagmus there was reason to believe that it had occurred at an earlier period; in 2 the eyes "had been rolled about," and in 2 others they had "twitched." Thus there was more or less evidence of nystagmus in 24 cases.

In one case (P.M. 23), in which trephining was done over the left lobe of the cerebellum, a few hours after the operation the left eye was strongly drawn down and affected with vertical nystagmus: next day this eye was not depressed, and both eyes showed slight vertical nystagmus. This resembles the effect noticed by Ferrier of electrical irritation of the cerebellar cortex in monkeys: "Frequently after the application of the electrodes a condition of nystagmus comes on, and lasts for some time" (Ferrier, *Functions of the Brain*, p. 191).

3. *Pupils.*—We found the pupils equal in 45 cases, unequal in only 7. It is possible that this inequality was physiological.

In 26 cases the pupils were of normal size (2 to 3½ mm.); in 25 cases the pupils were small; in 17 cases they were large.

In 23 cases we noted that the pupils responded normally to light; in 11 we found this reaction sluggish, and in 3 it tended to be of an oscillating character.

If the pupils are affected at all in the earlier part of the illness, they tend to contraction; at the later period, especially if hydrocephalus cause compression of the brain, they may become dilated and inert. On the whole, alterations in the pupils are less common than in tuberculous meningitis.

4. *Optic discs.*—We have notes of the condition of the optic discs in 42 cases, in some of which they were examined several times. In 27 cases they seemed to us to be quite normal. In 8 cases they were decidedly pale, but clear-edged, and in 3 of these there was also pallor round the disc or along the course of the retinal vessels. In 3 of these cases our opinion was confirmed by Mr. Marcus Gunn, and in 4 others by Mr. Donald Gunn. In 4 cases there was deficient clearness of edge, and in 3 distinct optic neuritis. Thus, out of 42 cases, there were only 7 which showed any evidence of inflammation of the optic papillæ, and in only 3 was this inflammation distinct.

This rarity of optic neuritis in posterior-basic meningitis is in marked contrast to its frequency in tuberculous meningitis, and is most remarkable; for the inflammation at the base of the brain extends forwards in many instances quite up to the optic commissure, which may be found surrounded by inflammatory lymph. This contrast seems to us to have a very important bearing on the yet unsolved problem of the causation of optic neuritis.

5. *Amaurosis.*—Although optic neuritis is rare, yet blindness, more or less complete, is not uncommon in posterior-basic meningitis. If the cornea of the infant be threatened by the finger of the observer, it will often be noticed that no winking occurs until the eyelashes or eyelids are actually touched. The light reflex of the pupil in these cases is often sluggish.

Not seldom the blindness is quite distinct, and has even attracted the notice of the mothers. They say that the child "takes no notice"; that is, that its eyes do not follow a moving object. This may continue some weeks, or even months, but it usually, perhaps invariably, passes off if the child survive.

Thus one child was said by its mother to have been "blind the last two weeks," and there was no winking when the cornea was menaced. A second at 6½ months after the onset of his illness "never notices," but nine months later he was thought to take notice. A third "did not notice" for some weeks. A fourth "did not notice at the time of head-retraction." A fifth, ten months after the onset, and presenting signs of hydrocephalus, was apparently blind. The mother "thinks its sight bad." Three months later we noted that there was no winking when the cornea was threatened, but that the eyes did follow to some extent, and that he would grasp a finger held in front of him. Two years after the onset his vision was again apparently normal. In a sixth case, three months after the onset, vision was very doubtful. "Doesn't blink. Looks towards the light, but mother thinks he does not grasp at things as he did five months ago." A month later (then twelve months old) the mother thought he recognised her at two yards' distance, but not beyond. A month later still the mother said that he could then recognise her across the room. After this he completely recovered. In a seventh case blindness was first noticed four weeks after the onset, and was still present when we last saw the child ten months later (then aged 1 year 9 months).

"Doesn't flinch when finger is close to eye. Doesn't follow with eyes, but follows sounds at once. Certainly blind for objects, but mother thinks there is sense of light." In an eighth case, "since the fit at onset (2½ years old), vision has apparently been lost." A ninth "lost sight after four weeks, and is still quite blind (twelve weeks after the onset)." In a tenth case, two months after the onset, the child "sees only light and darkness." Three and a half months after the onset Mr. Gunn's note was: "My impression is that she sees objects, such as the hand, when fairly close to her face." Five months after the onset we noted that she was "blind." The fontanelle was then distended and the sutures open. Four months later (at thirteen months old) the mother stated that the child could see a light; but we found that she did not follow a watch or a hand near her face, whereas she looked at once in the direction of a sound. In an eleventh case the child when first seen three months after the onset "took no notice." Three months later she was certainly blind, and it was thought that she had not even perception of light (hearing good). Six weeks later still she certainly had perception of light, and probably some vision of objects.

It seems possible that the amaurosis may be due to inhibition of the functions of the lower visual centres—the anterior corpora quadrigemina, corpora geniculata, and posterior parts of the optic thalami—caused by the inflammation in the subarachnoid space just above them, thus interfering with the conduction of visual impressions to the occipital cortex. This would not necessarily involve complete interruption of the light-reflex.

6. *Pseudoglioma.*—The peculiar destructive inflammation of the eye which has received this name from the similarity of the inflammatory mass to a gliomatous growth in the eye, we have once met with in a case of posterior-basic meningitis. The right eye showed ciliary injection. The iris was pushed forwards conically, and was motionless to atropine. The pupil was occupied by lymph. With the ophthalmoscope a white mass and one small vessel were seen. The left eye was normal. The right eye had been in the same condition since the beginning of her illness nine weeks ago; three weeks after an attack of measles from which she had, apparently, quite recovered. She became pale and cold, and looked as if she would have a fit; thirty-six hours later a fit occurred, followed by unconsciousness for several hours. She vomited much the next day, and repeatedly all the following week. The head became retracted on the day after the fit, along with the onset of vomiting. The retraction had remained unaltered for nine weeks, and during the last two weeks the head had increased in size.

Otorrhœa.—Since one possible method of production of basic meningitis is by extension of inflammation from the middle ear when otitis is present, probably due largely to spread of micro-organisms from the mouth, nose, and pharynx through the Eustachian tubes, it becomes of interest to ascertain how often a discharge from the ear occurs. We find that otorrhœa is a rare symptom, and in most of the instances in which it occurs is probably a mere coincidence. After the operation of paracentesis tympani it occurred three times, and in one or two others the operation revealed a small quantity of pus in the middle ear. But apart from operative interference otorrhœa occurred in only five cases. In one child there was discharge from both ears four months after the onset; it lasted only three days. In another, seven months after the onset, there was discharge of pus from the left ear; the child was fretful, costive, and vomited once, but the head was not retracted afresh. In a third, otorrhœa occurred "some months" after the subsidence of head-retraction. In all these instances the symptom was of no importance from the point of view of etiology. In a fourth case, however, so early as three weeks after the onset the left ear discharged thick yellow matter; the otorrhœa continued for five weeks; this may possibly have been started by an otitis which preceded the onset. In a fifth case otorrhœa was observed four weeks after the onset. Both tympanic membranes were punctured, muco-pus and some blood escaping. Three weeks later the mastoid antrum was opened and drained on each side: muco-pus and granulation tissue (P.M. 36).

On the other hand, we noted the absence of otorrhœa in 39 cases; and, as this is an obtrusive symptom, it was probably absent in the great majority of the cases in which we have no note on the point. It is, therefore, of rare occurrence. But its absence is not to be taken as evidence of the absence of otitis.

"Champing" movements of the lower jaw, lips, and tongue.—Such movements, simulating the actions of sucking and mastication, are not uncommon, and are sometimes very striking. They occur also in tuberculous meningitis. They are not confined to sucklings. We have seen them in children only three or four months old, but they were also frequent in a child aged one year and eight months; very marked and persistent for at least two months in a child aged two years, and present occasionally in a child aged five years. They may suggest to the parents the erroneous notion that the illness is caused by teething. Depression of the lower jaw and its return, often more or less rhythmical, is the movement most frequently noticed; but movements of the lips and tongue often accompany it. The mouth is usually kept closed meanwhile, but sometimes it is opened and shut frequently. Sometimes the tongue is protruded and retracted. Yawning is not uncommon. Grinding of the teeth sometimes occurs. It is possible that these movements may be the reflex result of a sensation of taste excited by irritation of the olfactory (and gustatory ?) centres situated in the region of the tip of the temporo-sphenoidal lobe. Several of the autopsies above reported revealed lymph or cicatricial thickening on the tips of these lobes. But it may also be due to direct irritation of the cortical motor centres for the tongue and lips.

Tonic spasm.—Tonic spasm is a very characteristic feature of posterior-basic meningitis. As affecting the muscles of the neck and giving rise to retraction of the head, it is present in practically every case of the disease; at all events, it would not be safe to diagnose meningitis of the posterior base if this symptom were absent, and in cases proved to be of this nature by autopsy it has almost always been present at some period of the clinical history; though we sometimes meet with cases of meningitis of the convexity, which has spread down to the posterior base, in which retraction of the head had been almost absent. Intense inflammation of the convexity may cause little or no retraction of the head if the posterior base be but slightly affected; while in cases of vertical meningitis there may be well-marked head-retraction if there is much lymph about the pons, medulla and vermiform process. The importance of this sign was pointed out twenty years ago, in a paper by Dr. Gee and one of us (Barlow) on "Cervical Opisthotonos of Infants," published in the *St. Bartholomew's Hospital Reports* for 1878; also in a paper on "Simple Basic Meningitis in Infants," read by one of us (Lees) before the St. Mary's Hospital Medical Society in 1882. Retraction of the head occasionally occurs in tuberculous meningitis also, but at a later period, with less intensity, and with much less persistence; it perhaps indicates the spread of the inflammation to the region of the posterior base, or to the anterior end of the superior vermiform process. But we have seen a few cases of tuberculous meningitis with persistent head-retraction, and have found in them an unusual amount of tuberculous deposit at the posterior base of the brain. The presence of this symptom is not, however, conclusive evidence of the existence of meningitis of the posterior base, for in P.M. 50, though the retraction of the head had been extreme and very persistent, yet hardly any lesion was found, except distension of the ventricles and subarachnoid spaces with watery fluid. Moreover, we have seen cases in which this symptom was relieved by puncture of the tympanic membranes so rapidly as to suggest that it is sometimes due mainly to otitis; but in almost all fatal cases autopsy reveals meningitis, present or past. Yet in one instance (P.M. 23) the region of the posterior base was quite healthy, while the tympanic cavities were full of viscid pus, extending on the left side into the antrum of the mastoid; and in another (P.M. 48) there was evidence of severe otitis on both sides, while the posterior base was normal; but there must have been inflammation in its immediate neighbourhood, for the fourth ventricle was obliterated by adhesions.

In the rare cases of head-retraction occurring in enteric fever, of which we have seen two examples, the immediate cause is probably otitis. In cases of pneumonia in children, head-retraction, when severe, is probably due to meningitis; but slight head-retraction may be caused by otitis without meningitis. In one case we have observed temporary retraction of the head at the onset of infantile paralysis.

Retraction of the head, even when very marked, is often unaccompanied by any other form of tonic spasm; the dorsal spine remaining straight and the limbs unaffected. But in some cases the muscles of the back and those of the upper and lower limbs become involved in the tonic spasm. The contraction of the dorsal muscles produces an opisthotonos, which may be more marked than that seen in tetanus. In one case it was so extreme that the occiput was brought within two inches of the buttocks, the body of the child being curved round backwards to an extraordinary degree. We have once seen the opisthotonos accompanied by left-sided pleurotonos.

Spasm in the limbs is usually present also, sometimes flexor, especially when transitory; but generally extensor when persistent. The extensor spasm is quite as marked and frequent in the upper limbs as in the lower, which is in special contrast to the position of the upper and lower limbs respectively in the late rigidity of hemiplegia. The upper limbs may be rigidly extended while the lower are flexed, or flexed while the lower are extended; or all four limbs may be extended, or be flexed. Sometimes the limbs on one side of the body are rigid, while those of the other side escape. Occasionally the spasm is limited to a single limb; thus one upper limb may be rigidly extended, while the other three limbs are nearly normal. When the tonic spasm of the upper limbs is moderate there is often some retraction of the arm at the shoulder, flexion of the elbow, partial flexion of the wrist with decided flexion of the fingers and thumb, and pronation of the forearm. In a higher degree of spasm the limb is adducted, rigidly extended, and superpronated, the fingers firmly

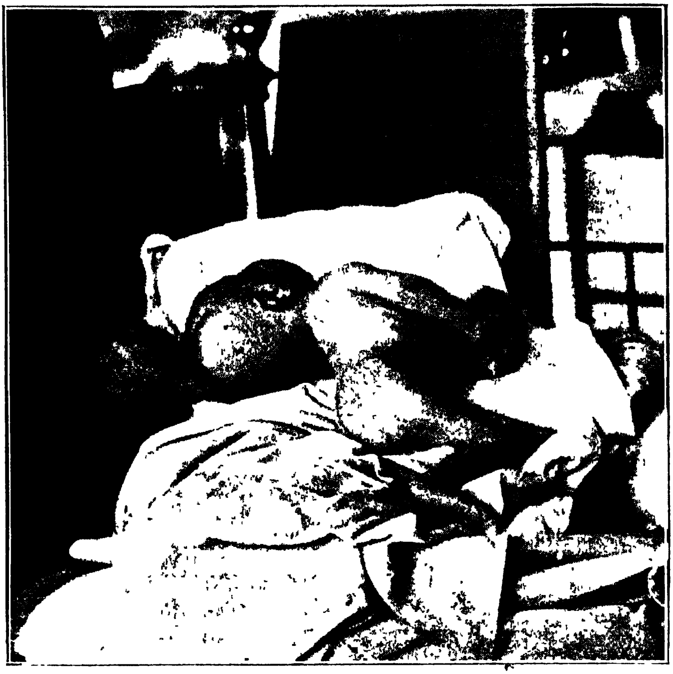

FIG. 28.—Marked head-retraction and rigid extension of right upper limb (P.M. 33).

clenched and pressing into the palm, the thumb either flexed or extended. One upper limb may be in this condition while the other is only slightly stiff, and still capable of being used in what appears to be partly voluntary movement. Fine tremors of the upper limbs are sometimes observed.

We have only once seen the hand in the tetany position; in that case the fingers were afterwards flexed, and finally the "claw-hand" was observed before death.

In moderate spasm of the lower limbs all the joints may be flexed, but in more extreme spasm the whole limb is rigidly extended, the heels drawn up, and the foot inverted. When the tonic spasm of the spinal muscles or of the limbs is marked, periodical exacerbations of the spasm are not uncommon, in which the back becomes more arched, the limbs more stiff, and sometimes also the chest rigid and the jaws closed, reminding the observer strongly of the phenomena of tetanus. And in other cases in which the tonic spasm is not permanent, there are recurring "fits" of tonic spasm in which the limbs and back become rigid, and sometimes (but not always) the head becomes more retracted.

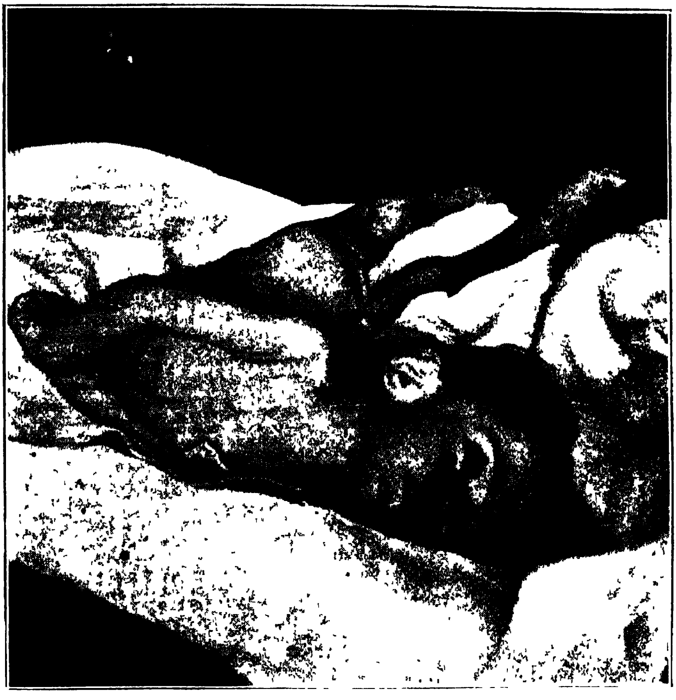
FIG 29.—Head-retraction, marked opisthotonos, rigid extension of limbs, superpronation (P.M 44).

There are thus, in posterior-basic meningitis, three characteristic forms of tonic spasm to be explained—retraction of the head, opisthotonos, extensor or flexor spasm of the limbs. With regard to these, three points should be noticed: first, that head-retraction is almost always present at some period of the illness, while tonic spasm of the back or limbs is much less common; secondly, that even when head-retraction is marked and persistent, the other two forms of tonic spasm may be quite absent; thirdly, that marked opisthotonos is usually associated with both retraction of the head and tonic spasm of the limbs, but that opisthotonos may be absent and head-retraction slight when there is much rigidity in one or more of the limbs.

It does not seem difficult to explain the head-retraction when there is inflammation at the posterior base, in the region of the lower part of the cerebellum and the medulla, just above the foramen magnum. Through the lower part of this space the first cervical spinal nerve passes outwards to escape from the spinal canal between the occipital bone and the posterior arch of the atlas. This nerve supplies the deepest muscles connecting the uppermost cervical vertebræ with the occipital bone—the recti and obliqui muscles and the complexus. Both by direct irritation of this nerve by the local inflammation and by reflex irritation of its nucleus of origin at the first cervical segment, a tonic contraction is caused in the muscles it supplies, and the head is drawn back. If the inflammation implicate also the second and third cervical nerves, or their nuclei of origin, the more superficial muscles of the same region, the splenius, trachelo-mastoid, and others, will become involved in the spasm, and finally the trapezius; the head-retraction will then be intense.

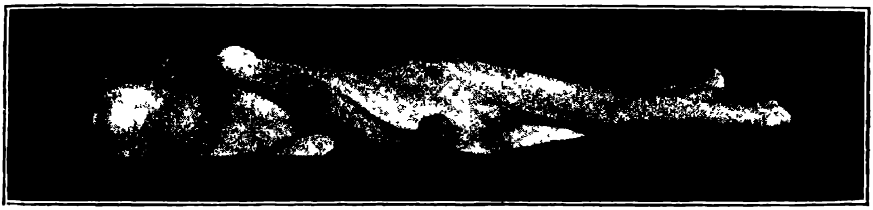

FIG. 30.—Head-retraction, rigid extension of limbs, but no opisthotonos (P.M. 50).

A similar reflex spasm may doubtless be produced by irritation of other important structures in the near neighbourhood. Thus Prof. Ferrier (5) found that the application of a weak induced current to the anterior extremity of the superior vermiform process caused head-retraction, with upward movement of the eyes, a tendency to extension of the legs, and some spasmodic movements of the arms. Similar phenomena and even opisthotonos were still more readily produced when the electrodes were applied to the corpora quadrigemina. Thus it is obvious that a focus of inflammation in the subarachnoid space overlying the corpora quadrigemina, and just in front of the superior vermiform process, will readily produce head-retraction, even though the cerebello-medullary cistern be free from inflammation. And the connections of the auditory nerve with the medulla and the restiform bodies make it easy to imagine

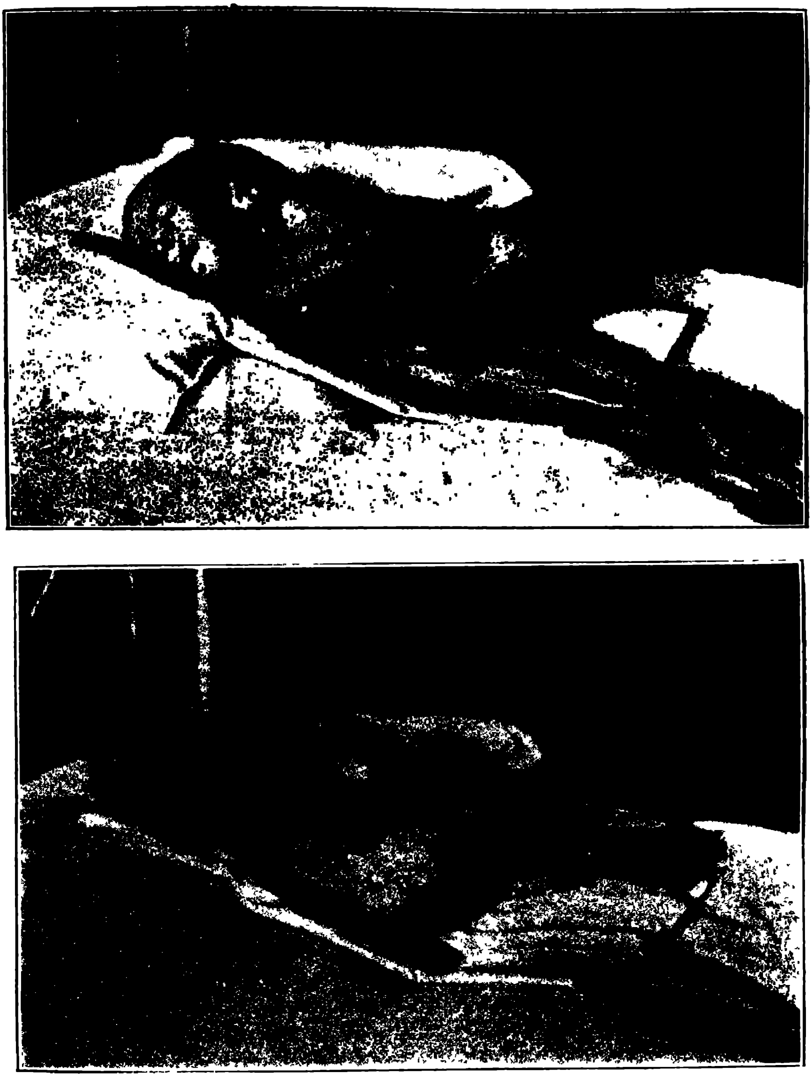

Fig. 31.—Moderate head-retraction, flexor spasm of limbs.

how abnormal conditions of the auditory apparatus may excite a reflex spasm of the muscles which retract the head, without any meningitis at all.

The much less frequent occurrence of opisthotonos, and its association with rigidity of the limbs, seem to point to a different mechanism. The opisthotonos is not due to spinal meningitis, for we have seen it very well marked in a case where the spinal cord was found normal at the necropsy (P.M. 31). Some of the post-mortem evidence already stated suggests that opisthotonos and tonic spasm of the limbs may be due to irritation of the cerebellum. In two of the six cases in which one or both of these phenomena were most marked, we were unable to obtain permission for an autopsy. In two others (P.M. 26 and 40) we found extremely dense adhesions at the posterior base, firmly uniting the cerebellum to the medulla and to the dura mater, and so to the bone at the foramen magnum; in one of these the cerebellum was hollowed out by fluid pressure. In a fifth, however (P.M. 44), the only evidence of any special pressure on the cerebellum was that the fourth ventricle was much dilated, and in a sixth (P.M. 50) little was discovered except distension of the ventricular cavities and subarachnoid space. Yet the fact that the two cases in which the cerebellar adhesions were the most extensive and the firmest, were two of those in which opisthotonos or tonic spasm of the limbs was most extreme, seems to suggest that the cerebellum may have been the source of these special developments of tonic spasm. No lesion of the cerebrum is known to produce opisthotonos or persistent tonic extensor spasm of the upper limbs; the late rigidity following hemiplegia is in the upper limbs always of flexor type. We have already pointed out the likeness of these forms of tonic spasm, with their remarkable temporary exacerbations, to the phenomena of tetanus, in which disease the early and marked affection of the motor division of the fifth nerve, with the accompanying rigidity of the facial muscles and the occasional paralysis of the seventh nerve, indicates clearly that the main seat of morbid action is in the pons, with which the cerebellum has such intimate connections. It may here be noticed that nystagmus and contraction of pupils, which we have shown to be frequent symptoms in posterior-basic meningitis, were observed by Ferrier when the cerebellar cortex was stimulated by a weak induced current. Hence we would suggest that the nystagmus and the contracted pupils observed in posterior-basic meningitis are due to excitation of the superficial gray matter of the cerebellum by the local inflammation; and that in the severer cases, in which the cerebellum is more profoundly affected, opisthotonos is caused by more intense irritation of its middle lobe, while tonic spasm in the limbs results from more intense irritation of the lateral lobes.

If these suggestions are correct, the phenomena observed in posterior-basic meningitis seem to throw some light on the normal functions of the cerebellum. For the tonic rigidity already described can only be an exaggeration of a normal action. If the cerebellum, irritated by disease, is capable of producing these tonic spasms, it is in the highest degree probable that in health it exercises a constant but less marked influence of a tonic kind on the muscles of the back and of the limbs. For the dorsal muscles a tonic co-ordinating influence is required in the acts of sitting, standing, and walking; it probably emanates from the central lobe of the cerebellum, and especially perhaps from the large and bilaterally associated "roof-nuclei."

With regard to the limbs, Dr. Risien Russell has shown that, in dogs, ablation of one lateral lobe of the cerebellum produces at first rigid extension of the anterior limb of the side of the lesion, a similar but much less marked spasm of the posterior limb, and very slight spasm of the opposite posterior limb. The knee-jerk on the same side is exaggerated, that on the other side is for a short time lessened, afterwards exaggerated. There is also for a time paresis and some anæsthesia of the posterior extremities, and to a slighter degree of the anterior extremity on the same side as that of the cerebellar lesion. From these experimental observations it seems to follow that in some way or other the lateral lobe of the cerebellum exercises normally a tonic influence on the muscles of the limbs on the same side. It is possible that this may be exerted indirectly through the motor cortex of the opposite hemisphere; but it seems more likely that the rigidity of the limbs observed in extreme cases of posterior-basic meningitis is the result of a direct downward effect on the anterior-horn cells of the same side of the cord, the "cerebellar influx" of Dr. Hughlings Jackson.

Clonic spasm.—Convulsions (more or less general clonic spasm) were noted as having occurred at some period of the illness in 54 cases, and in 26 of these they were the initial symptom. But we satisfied ourselves that many of the spasmodic attacks called by the parents "convulsions" were really paroxysms of tonic contraction such as we have described. These paroxysms are often accompanied by violent screaming. Yet it is certain that general epileptoid convulsions may occur in the early stage of both vertical and posterior-basic meningitis; in both they are doubtless of cortical origin. For even in posterior-basic meningitis there is often post-mortem evidence of slight inflammatory changes in the membranes covering the convolutions. In vertical meningitis the noteworthy fact that convulsions are not present in every case may perhaps be explained by the supposition that the cortical cells are paralysed or inhibited by the toxines produced by the microbic growth.

In some cases convulsions are frequently repeated at the onset of the disease. At a later period convulsions are much rarer, and may be isolated. Sometimes they occur at the very end, not seldom with a rising temperature, occasionally with hyperpyrexia.

There may also be clonic spasms affecting the eyelids, the face, hands, or fingers, or two or more of these. In some cases the head is jerked forwards; in others the trunk is jerked forwards, as by a momentary opisthotonos.

Paralyses.—Paralytic symptoms are occasionally met with in posterior-basic meningitis, though they are much less common than spasm. The motor nerves of the eyeball are rarely, if ever, paralysed, either in vertical or in posterior-basic meningitis.

In 5 cases we have seen paresis of the facial nerve on one side, involving both upper and lower divisions of the nerve. In one of these we tested the electrical reactions, and found distinct loss of response to faradism, with slight increase in the response to galvanism, as compared with the unaffected side. In none of the 5 was there otorrhœa or any indication of disease of the petrous bone. It may fairly be inferred that the paresis was the result of the implication of the facial nerve in the meningeal inflammation.

Inability to hold the head erect is often observed during recovery from posterior-basic meningitis. There may also be inability to sit up.

Paralysis of the limbs is rarely observed. In one case (age 3 months) the onset was by convulsions, after which the head was retracted, and there was complete palsy of the left arm and hand. The latter symptom passed away in about six weeks; its method of recovery was the reverse of that of an ordinary hemiplegia, the distal parts recovering before the proximal. Movements of the fingers were observed by the mother a fortnight after the occurrence of the paralysis, movements of the hand ten days later. When we first saw the child, a month after the onset, it could move the hand and fingers but not the arm. There was moderate retraction of the head, some alternating squint, and some coarse nystagmus. Three weeks later there were distinct indications of hydrocephalus, but the child could now move its left arm as well as the fingers and hand; the palsy had disappeared. Seven months later slight weakness of the left side of the face was observed. At a year old it was still unable to sit up without support.

Crying and screaming.—Screaming is often observed at the onset of the meningitis. It may continue many hours—"violently all day," or "during the whole of the first night." It is then sometimes associated with convulsions. It is always increased in violence if the child be in any way disturbed. When not moved the child may be quiet, but when it is lifted or turned, or its head is raised, sometimes even "at the least noise," the screams break out afresh. The screaming is suggestive of severe pain; but it is well to remember that Ferrier found (5) that even slight irritation of the posterior corpora quadrigemina always produced an outcry, "the utterance of a short bark or cry on the slightest contact of the electrodes, and every variety of vocalisation when the stimulation is continued." In other cases the pain appears to be less acute, but there is "much crying at first," or the child is "very fretful"; in other cases, again, the symptom is completely absent. When present, it usually diminishes as time goes on, but sometimes it may recur at intervals for several weeks. In one instance it was "marked throughout" an illness of seven weeks; in another it was "marked from the beginning, and still present three months after the onset." A child, aged 3 years, "screamed when approached four weeks after the onset." It sometimes, but by no means always, accompanies the tetaniform tonic spasms. In one infant of five months, with chronic extensor spasm of the right upper limb, the character of the cry was very remarkable; it consisted of an occasional loud squeal, prolonged and rising in pitch, reminding us of the peculiar sound produced by a fog-siren.

Screaming may be present at the onset only. Thus one boy, three days after the onset by vomiting, screamed every five minutes for one hour, and his head became retracted, but there was no return of the screaming throughout the case.

In feebler children there may be merely a whining moan, more or less frequent.

During the hydrocephalic condition, which is so apt to follow the acute disease, there is little or no crying. Thus a child "was very fretful during the first fortnight, but now the head is enlarging he seems well and cheerful." Infants suffering from chronic hydrocephalus, whether congenital in origin or the result of basic meningitis, do not emit sudden outcries of pain. To this condition the term "hydrocephalic cry" is quite inapplicable. Perhaps the reason for this is that the increase of intracranial pressure is very gradual, and is diminished by the yielding of the sutures and enlargement of the skull.

Respiration.—The catarrh which frequently precedes the onset of posterior-basic meningitis may involve the bronchial tubes and the air-cells, causing bronchitis and broncho-pneumonia; in this case the breathing will be more rapid than normal, but it will nevertheless remain fairly regular, exhibiting nothing more than the slight irregularity which is common in infants. At a later stage of the illness cerebral abnormalities of respiration are apt to show themselves. Occasional deep sighing inspirations may be observed. These are sometimes, but not commonly, associated with yawning. The sighing respiration may gradually pass into markedly irregular breathing, but the irregularity is often rhythmical. "Cheyne-Stokes" breathing is sometimes seen; thus in one infant of seven months there was a long slow wave of increasing and diminishing inspirations, about nine in all, followed by a pause; sighing was also present. Both tympanic membranes were punctured, and two days later the Cheyne-Stokes rhythm was hardly noticeable. In another case we noted "breathing in groups, much up and down." But we have more frequently noticed a different type of cerebral breathing. It consists of a number —from two to six—of equal deep inspirations, followed by a pause of several seconds' duration. Thus an infant of 4 months old (observed in 1878) "takes three or occasionally four deep rather noisy inspirations in quick succession, occupying five seconds, then in the following ten seconds there is no breathing at all." A week later we found "the breathing in cycles still, two deep inspirations, then ten seconds' interval." This child took large doses of iodide of potassium (3 grains every two hours), and made an apparent recovery; but it died unexpectedly three months later, and was then found to have hydrocephalus (P.M. 47). In another child (in 1879) the periods consisted of three deep inspirations followed by a pause of twelve seconds. This child had "sighed a great deal" during the earlier part of its illness; it was also observed to yawn much. In an infant of 10 months (in 1882) three deep inspirations, occupying seven seconds, were followed by a pause of seven seconds. Here also much sighing had been present previously. In an infant of 5 months (in 1885) five or six respirations were followed by a pause equal in duration to three respirations, then came a deep sighing inspiration. In another infant of 5 months (in 1887) the respirations were "in a series of three or of four, followed by a long pause, at the end of which a sigh often commences a fresh set."

This type of cerebral breathing differs from the "Cheyne-Stokes" in the absence of the ascending and descending rhythm, the grouped inspirations being of equal or nearly equal depth. We have used the term "cyclical breathing" to distinguish this symptom, though we recognise its imperfection, for the Cheyne-Stokes breathing is also in cycles.

Shortly before death the respiration may be exceedingly irregular; in an infant 5 months old the number of respirations varied from eleven to fifteen in fifteen seconds, and at very unequal intervals; the heart's action was also very rapid, about 240 (temperature 102°·9).

When tonic spasm of the back or limbs is present, the muscles of the thorax may become rigid and impede the respiration, as in tetanus.

Pulse.—The frequency of the pulse in infants suffering from meningitis may be quite normal, or it may be much increased. In infants of not more than 12 months old we have often found a pulse-rate of 120 (which may be considered normal for the age), occasionally 130, 140, 160, 170, 200, 220, and even 240. We have not found a slow pulse in children of this age; perhaps this indicates that the inhibitory control of the heart by the vagus is in these young children imperfectly developed.

With regard to older children, a girl aged 1 year and 8 months had a pulse of 70 five days after the onset, of 110 (normal rate for her age) eleven days after the onset, and of 200 in the final convulsion, during which the temperature rose to 106°. A child aged 2 years and 4 months had, at various periods, pulse-rates of 108, 150, 140, and 180. A child aged 3 years and 9 months had, in the eighth week of her illness, a pulse of 96 "with four intermissions in a quarter of a minute." Previously it had been regular, and at a rate of 128. In the tenth week it was again 128, feeble and regular. A child aged 5 years, whose normal pulse-rate should have been about 100, gave in five observations the following numbers—56, 82, 120, 84, 76. Here we see clearly the inhibitory action of the vagus on the heart.

Vaso-motor phenomena.—Spontaneous flushing is sometimes very marked in cases of meningitis, but the symptom is by no means so frequent, either in the vertical or posterior-basic class, as in tuberculous meningitis; in some it seems to be entirely absent. Sometimes hyperæmic areas of skin are produced where pressure has been applied. The phenomenon usually, but very incorrectly, described as "tache cérébrale" is sometimes quite distinct, but often absent.

Temperature.—In the rapidly fatal course of vertical meningitis the temperature often rises quickly, attaining 105° or more. In one case, illness only forty-seven hours, it was 107°. In a second case, illness six days, the temperature was never lower than 101°, and it rose to 106° just before death. In a third case, illness ten days, it was 105° on the second day, 104° on the third, 103° on the fourth, 101° on the fifth, and 100°·6 on the sixth. On the ninth day it was only 99°.

In the less violent onset of posterior-basic meningitis the pyrexia is more moderate unless broncho-pneumonia is present also. In one case which ended in recovery, the temperature at first was on more than one occasion 106°, but this was explained by dull patches in the lungs. If there be no pneumonia, the pyrexia, even at the onset, is usually slight. Afterwards the temperature is prone to be very irregular. Thus in one case during the first week it ranged from 98° to 101°; in the second week it was nearly normal.; in the third week quite normal; in the fourth week 98° to 100°; in the fifth week 102°; and on the day of death 106°·5. In another case the temperature in the fourth week varied from 98° to 100°, with a rise to 102°·6 for one day (with marked increase of vomiting); in the fifth week the average was lower, and maximum 100°; in the sixth week mostly subnormal (but with one rise to 100°); in the seventh week subnormal, but a sudden rise (with a convulsion) to 107°·2 occurred twenty-four hours before death, then a fall to 102°·5, and a rise to 103°·2.

Terminal hyperpyrexia is not uncommon; we have notes of its occurrence in seven cases: the two highest recorded temperatures were 107°·8 and 108°.

In the cases which last for many weeks the temperature becomes normal or subnormal; before death it may be as low as 96° or even 95°, taken in the rectum: in one instance it was 95° for three days, and 88°·8 just before death.

Abdomen.—In many cases the abdomen is normal in appearance; in a few it is more or less retracted. The extreme retraction so characteristic of tuberculous meningitis we have never seen in posterior-basic meningitis.

Vomiting is a frequent, almost an invariable, symptom. It nearly always occurs at or soon after the onset, and is then sometimes severe. Subsequently it may occur once or twice daily for three or four weeks together; or in periods of a day or two, during which it may be incessant.

Bowels.—In about half the cases the frequency of defæcation was normal. In some instances there was a little diarrhœa, especially from intestinal catarrh at the onset. In the later stages of the disease there is greater tendency to constipation, but it is rarely very pronounced. Constipation is not nearly so frequently present as in tuberculous meningitis.

Urine.—In the cases in which we have examined the urine there has been nothing abnormal.

Skin.—In most cases no eruption is observed, but in a few an ill-defined erythema occurs. Herpes is very rare in this disease, which is noteworthy considering its frequency in the epidemic form.

Reflexes.—The knee-jerks are probably always present, and often increased. The plantar reflexes are sometimes feeble.

Joints.—In rare cases, especially of the acute vertical class, one or more joints may be affected, suggesting a pyæmic state; and in three instances we have found after death small purulent collections in joints or tendon-sheaths. In the more chronic posterior-basic cases we have occasionally observed slight but definite thickening over the region of the junction of the shaft and upper epiphysis of the humerus; also in the neighbourhood of the tendon-sheaths on the back of the wrist. It is remarkable that in the progress of the disease this thickening may so far subside as to be no longer clinically distinguishable.

Wasting.—Marked wasting is often a feature of meningitis, especially in the chronic cases. Sometimes it may be accounted for by persistent vomiting, occasionally by diarrhœa; but it is often greater than the amount of vomiting will explain, and sometimes it is extreme when there has been hardly any vomiting and no diarrhœa, and even when the child has been carefully fed and tended in hospital. Thus, a child aged two years, admitted into hospital a fortnight after the onset of his illness, and then described as "plump," though he was carefully fed and had no vomiting or diarrhœa during the eight weeks he was in the hospital, wasted steadily, and at death was in a condition of extreme emaciation. Children which recover often remain emaciated for many weeks after the subsidence of all active symptoms.

Hydrocephalus.—The post-mortem evidence described in this article proves that distension of the cerebral ventricles with fluid is a very frequent result of posterior-basic, though not of vertical meningitis. The character of this fluid varies. Sometimes it is turbid, containing floating fibrinous flakes, and in the lower parts of the ventricles what may fairly be called pus. Fluid of this description is obviously in part of inflammatory origin. In other cases the fluid which distends the ventricles is nearly transparent, is of very low specific gravity, and contains but a very small amount of albumin. When this is the character of the fluid, some obstruction will almost invariably be found in the narrow passages by which the ventricles are connected with each other and with the subarachnoid space;—at the exits from the fourth ventricle, in the iter, in the fourth ventricle itself, in the posterior subarachnoid space, around the upper cervical cord; rarely at the foramen of Monro. Here the hydrocephalus is due to retention of fluid normally secreted. This distinction will be made evident by the following analyses of ventricular fluid from two cases of posterior-basic meningitis under our care; the analyses were made by Dr. Vaughan Harley.

In the first case the fluid, removed by aspiration from the lateral ventricle during life, was of a pale straw colour, containing some deposit of fibrin; its specific gravity was 1009. On analysis Dr. Harley found:—

Water	97·256 per cent.
Total solids	2·744 ,,
(Proteids 1·426)	
	100

This result he contrasts with the analysis of cerebro-spinal fluid from a spina bifida obtained by Hoppe-Seyler, as follows:—

Water	98·749 per cent.
Total solids	1·251 „
(Proteids 0·162)	
	100

Dr. Harley points out that the higher specific gravity of the fluid analysed by him, and the larger quantity of proteids present in it, indicate that it was, partly at all events, of inflammatory origin. But the fact that the specific gravity is still low seems to suggest that, though it contained inflammatory products, it consisted mainly of cerebro-spinal fluid.

FIG. 82.—Hydrocephalus with distension of ventricles caused by adhesions closing the foramen of Majendie. (An incision has been made through the valve of Vieussens, to expose the interior of the fourth ventricle.) No recent lymph. Symptoms of meningitis six months before death.

In the second case Dr. Harley's analysis gave the following result: specific gravity, 1003; water, 98·895 per cent; solids, 1·105 per cent. This was therefore pure cerebro-spinal fluid, and in this case the hydrocephalus must have been due to simple accumulation caused by obstruction. Though the obstruction had been caused by inflammation, the hydrocephalic fluid was not of inflammatory nature.

In a case of posterior-basic meningitis, recently under our care at the Hospital for Sick Children (P.M. 46), the cerebro-spinal and lateral foramina were closed, and the ventricles were greatly distended with clear fluid. Ten ounces of this were collected, and about four ounces more escaped. The specific gravity was 1010. Cold nitric acid gave a slight cloud. After concentration by evaporation, a distinct copper-reducing action was obtained. This reaction is very characteristic of cerebro-spinal fluid: it is not due to sugar, as was formerly thought, but probably to pyrocatechin ($C_6H_6O_2$) (7). In the presence of this substance and in the peculiarity of the proteids (globulin, albumose, occasionally peptone, rarely albumin, never fibrinogen) cerebro-spinal fluid differs, according to Dr. Halliburton, from the fluids of the serous cavities, which "may be briefly described as diluted blood-plasma," and are, "like normal lymph, transudations from the blood." He therefore concludes that cerebro-spinal fluid is not a transudation but a secretion. How is this secretion produced? We would suggest that the choroid plexuses are an apparatus for filtering off watery fluid into the ventricles, analogous to the glomeruli of the kidney which filter off watery fluid into the renal tubules. Each of the four ventricles is provided with a choroid plexus of its own. Such a plexus consists of a mass of blood-vessels freely supplied with arterial blood. Those of the lateral ventricles, which are by far the largest, have a double arterial supply to each, from the internal carotid and from the terminal branches of the basilar. This endowment seems to imply a function of special importance. Again, the epithelium of the ventricles "changes its character where it covers the plexuses. It is there composed of large spheroidal corpuscles, in each of which is seen, besides a distinct nucleus, several yellowish granules, and one or more dark round oil-drops" (13). This must surely be a secreting epithelium. It is probable that by means of the choroid plexuses a constant secretion of watery fluid into the ventricles is kept up, in order to maintain the water-bed on which the brain rests and the water-cushion which surrounds the cord within the spinal theca. One of our cases (P.M. 48) suggests that when the choroid plexuses are much injured, or compressed by inflammation, the flow of cerebro-spinal fluid may be considerably diminished. This patient recovered temporarily from an attack of posterior-basic meningitis and seemed in good health for seven months, though the fourth ventricle had become obliterated and hydrocephalus had resulted; the choroid plexuses were found to be cystic. If the cerebro-spinal fluid is being constantly secreted, it is necessary that it should be as steadily drained away and absorbed. The ventricular cavities communicate with the general subarachnoid space by means of the cerebro-spinal foramen and the two lateral foramina of the fourth ventricle, and by these alone. "Injections into the subarachnoid space of the cord," say Key and Retzius, "produce complete filling of the interior of the whole velum interpositum quite up to the lateral choroid plexuses which course at its edge in the lateral ventricles, but the injection never penetrates into the choroidal tufts themselves, either here or in the median plexus of the third ventricle." There are therefore no issues from the lateral ventricles into the subarachnoid tissue within the velum interpositum; and it is still doubtful whether the cleft in the pia mater along the descending horns, described by Merkel and Mierzejewsky, really exists (14).

Thus the only escape for fluid from the lateral and third ventricles is through the iter, the fourth ventricle, and its three openings into the posterior subarachnoid space, and so into the meshwork of subarachnoid tissue extending under the base of the brain, and into the large subarachnoid space surrounding the spinal cord in its whole length. Hence it exudes along the lymphatic sheaths of all the outgoing nerves, both cerebral and spinal. The small diameter of the passage connecting the third and fourth ventricles ensures that the flow of fluid from the lateral ventricles to the base of the brain shall not be too rapid. Thus there is provision for a steady, slow, and effectual drainage of the fluid secreted by the choroid plexuses. The necessity for this drainage is probably greater in infancy, while the still developing brain demands a more active blood-supply, in which the choroid plexuses will participate, than in later life. The cerebro-spinal foramen is much larger during the first year of life than it is afterwards, as may be seen in the accurate drawings appended to the first of the lectures on "Rest and Pain" delivered by Mr. Hilton thirty years ago; we may add that in his second lecture he gave an excellent illustration of the closure of this foramen by cicatricial membrane, and described the hydrocephalus which resulted. In adults the foramen is very small, "about the size of a pin's head, sometimes larger," as was pointed out by our colleague Dr. Dickinson. In the horse the cerebro-spinal foramen is normally closed, and this fact has been used as an argument against the dependence of hydrocephalus on obstruction of this foramen; but Key and Retzius have demonstrated that in this animal, though no median opening of the fourth ventricle exists, the two lateral openings are large.

We may here add that cerebro-spinal fluid, as obtained from any point below the fourth ventricle, cannot be an absolutely pure secretion; it must contain waste products resulting from cerebral metabolism, for into the subarachnoid space surrounding the brain open the lymphatic sheaths of all the cerebral arteries, and Dr. Bevan Lewis has shown that the nerve-cells of the brain are placed within "pericellular sacs," each of which has a definite lymphatic connection with the wall of a small blood-vessel. Thus the fluid obtained from a spina bifida must contain a small amount of waste products from the brain; but the alteration in its chemical composition thus produced will be but trifling if the secretion of fluid by the choroid plexuses is active and constant. This explains why the fluid obtained from a spina bifida by Hoppe-Seyler contained a slightly greater percentage of solids and a slightly smaller percentage of water than the second specimen of hydrocephalic fluid analysed by Dr. Harley, and seems to prove that the latter was the pure secretion of the choroid plexuses.

It is impossible for any absorption of cerebro-spinal fluid to occur in the choroid plexuses, for their free arterial supply must keep the blood in these vessels under high pressure; and to whatever extent the pressure of the ventricular fluid may increase in chronic hydrocephalus, it can never equal that in the arterioles, or the cerebral circulation would cease. To us it seems as absurd to suggest that the choroid plexuses absorb as well as secrete, as it would be to make the same suggestion with regard to the glomeruli of the kidney.

Nor is it likely that much, if any, absorption of cerebro-spinal fluid is effected by the veins. While the arterial plexuses project into the ventricles and are merely covered with a layer of modified epithelium, the veins which receive the blood from these plexuses—the "veins of Galen"—are withdrawn from the ventricles into the interior of the velum interpositum. Even where this structure forms the roof of the third ventricle the ependyma is still continuous beneath, and it seems unlikely that any great amount of transudation occurs through it. We have only once found evidence of compression or thrombosis of the veins of Galen, and in that case (P.M. 40) extensive adhesions, obliterating the drainage-channels, existed also. We believe that the current explanation of the production of hydrocephalus by obstruction of these veins has no basis of fact. It is of course possible for a tumour to press on these veins, but such a tumour will almost inevitably compress the iter or the fourth ventricle; in this case hydrocephalus will certainly be produced. Similarly compression or thrombosis of cerebral sinuses does not produce distension of the ventricles unless the drainage-channels be interfered with. We may add that, if hydrocephalic fluid were a cerebral dropsy resulting from venous obstruction, it ought to contain a larger amount of albumin.

There is, however, another anatomical arrangement by means of which excess of fluid in the subarachnoid space surrounding the brain may escape, and that is through the arachnoidal villous processes, figured by Key and Retzius, which project into the sinuses of the dura mater, especially the longitudinal, in great numbers, but which only attract attention when much hypertrophied, being then known as "Pacchionian bodies." Key and Retzius have demonstrated, by means of injections of coloured fluids into the subdural and subarachnoid spaces respectively of the spinal cord, that each of these spaces communicates in the arachnoidal villi with the sinuses of the dura mater, and that fluids thus injected escape into the blood-current within these sinuses. These arachnoidal villi exist in early life, but they are apparently less developed than at a later period; and it is possible that they are physiologically of minor importance in infancy. Probably their efficient action is interfered with, when the ventricles become distended, by the resulting pressure on the cortex of the brain and its membranes.

As further evidence of the production of chronic hydrocephalus by obstruction of the drainage-channel, we add brief notes of two cases in which it was caused by blocking of the cavity of the fourth ventricle by a tumour of the middle lobe of the cerebellum.

CASE I.—Emily B., aged 8 years, fell and struck the back of her head against a form, seven weeks before her admission into hospital. Headache came on next day and had continued ever since; it was frontal (not occipital). No vomiting or constipation. Cannot now walk or even sit up. Sight gradually impaired; blind last ten days. On admission, no paralysis of limbs or face. Marked optic neuritis in both eyes. Pupils rather dilated, quite motionless to light or darkness, but act readily with accommodation. Frontal headache. Intelligence good. While in the hospital convergent strabismus was seen; pulse irregular at times; motions and urine passed in bed. No vomiting. On the nineteenth day after her admission, her breathing became slow and her face blue; finally the respiration stopped and she was cyanosed, but the heart was still acting. Artificial respiration kept her alive for an hour, and improved her colour and her pulse; but when it was given up she died.

P.M.—Convolutions flattened. Lateral ventricles distended with clear fluid. Foramen of Monro, third ventricle, and iter all distended. Iter admitted a lead-pencil. Valve of Vieussens thin, almost membranous. A tumour, size of an apricot, occupied the lower part of the middle lobe of the cerebellum, and encroached equally on the two lateral lobes. It was spherical, gelatinous-looking on section, with some yellowish spots in its centre, and a distinct border. It grew into the fourth ventricle, filling it completely, and pressing out the anterior wall of the ventricle into a concave surface fitting over the round tumour. It did not reach the posterior surface of the middle cerebellar lobe. Cerebro-spinal foramen quite obstructed internally. No meningitis. No tubercle anywhere. Viscera normal.

CASE II.—Rose C., aged 5 years, fell down an area four months before her admission into hospital. On admission has headache. Cannot stand; sways and falls, usually backwards. Pupils act to light. Knee-jerks absent. Slow nystagmus when she looks to either side. Mental faculties seem defective. Optic discs normal. Two months later optic neuritis appeared in both eyes, and there was vomiting for four days. A month later still swallowing was difficult, and there was some paresis of right side of face. She gradually became very lethargic, with widely-dilated and motionless pupils, and was for several weeks before her death only semi-conscious, with convergent strabismus.

P.M.—Convolutions flattened. Lateral ventricles much distended with clear fluid. Corpus callosum very thin. Third ventricle dilated. Iter somewhat dilated. Cranial ganglia and nerves normal. Lateral lobes of cerebellum normal. Middle lobe almost entirely replaced by a tumour about as large as a Maltese orange, probably gliomatous. The tumour had grown forwards, and quite obliterated the cavity of the fourth ventricle, having become adherent to its floor, but it could be separated from it without much difficulty. It extended as far forwards as the valve of Vieussens, but had not affected the fourth nerves or the corpora quadrigemina. The posterior surface of the middle lobe was normal, but the tumour was immediately beneath. Cerebro-spinal foramen open and normal. No meningitis. An anterior mediastinal gland, of the size of an almond, was apparently infiltrated with new growth; its section was gelatinous-looking, not caseous. No other glands affected. Viscera normal.

In posterior-basic meningitis a study of the post-mortem results seems to afford absolute proof that the accumulation of fluid is due to obstruction of the drainage-channel. When the obstruction closes the issues from the fourth ventricle, or fills the posterior subarachnoid space, or glues together the spinal membranes, the fourth ventricle is distended along with all the others. (In many cases we have found also obstruction of the iter; and in P.M. 44, closure of the foramen of Monro in addition.) Where the obstruction is only at the iter, as in P.M. 14, 22, and 31, or the cavity of the fourth ventricle is obliterated by adhesions, as in P.M. 48, the distension affects the lateral and third ventricles only, the fourth being not dilated. In P.M. 36 the lower part of the fourth ventricle was obliterated by adhesion and the upper part distended, with the iter and other ventricles, by clear fluid. But the lateral ventricles never escape distension when the fourth is distended, which depends upon the fact that the only escape for fluid in the lateral ventricles is through the fourth into the subarachnoid space. When more than one obstruction exists, the fluids in the cysts thus formed may differ in character, as in P.M. 33. In the subarachnoid space of the cord localised collections of fluid may be found, limited by adhesions of the spinal membranes.

In some cases of *chronic hydrocephalus*, which at first sight seem to be of *congenital* origin, careful inquiry elicits a history of symptoms pointing to posterior-basic meningitis at a very early period of infancy. Thus:—

Joseph F., aged 12 months, has marked hydrocephalus. The circumference of his head measures 22 in., over the vertex from meatus to meatus measures 15¾ in.; the anterior fontanelle is very large. When three months old he had an illness, in which his head was drawn back and he squinted; he cried much for three weeks. Three weeks later it was observed that his head had begun to enlarge. The enlargement has steadily increased.

Herbert S., aged 17 months, has a very large head (measurements 22½ and 15½). When six months old he cried much for a fortnight, and seemed to have pain at the back of his neck. The head was drawn back, and he screamed if a hand was put under his neck to raise him, or if the head was moved in any way. After this the head grew larger every week until he was fourteen months old.

George A., 10 months old, has a large head (measurements 20⅛ and 14), and has only just begun to put his hand to his mouth. Nystagmus is present. He was an eight months' child. At one week old he had jaundice and frequent general convulsions. At three weeks old he would not allow his head to be raised, "always wanted to be lying." At about two months old it was observed that his head was too large, and it has progressively increased in size.

Even in cases of hydrocephalus undoubtedly congenital there is reason to believe that some are due to meningitis occurring during intra-uterine life. For instance:—

Ellen R. was found to have too large a head when only two weeks old. At two months old the fontanelle and sutures were distended, and the measurements were 18½ and 12 in. There was much atrophy of the choroid in both eyes. At 4½ months old 4 oz. of fluid were withdrawn by aspiration; it was watery, transparent, of specific gravity 1010, and contained albumin. Three weeks later she died from diarrhœa. P.M.—No fluid in subdural space. Large accumulation of fluid and only a very thin layer of brain-substance beneath the membranes; corpus callosum absent. When the fluid was removed the basal ganglia were seen, and the third ventricle somewhat dilated. The iter was pervious. The fourth ventricle was twice its ordinary size. Immediately below the fourth ventricle the overlapping edge of the cerebellum was firmly united by fibrous adhesions to the medulla oblongata. It is highly probable that these adhesions were of inflammatory origin, and that the inflammation occurred during intra-uterine life.

Florence O., at three months old, had a very large head, nystagmus, and large patches of choroidal atrophy in both eyes. She died at six months old, the circumference of the head being then 25 inches. P.M.—The head contained 98 ounces of clear fluid, partly above the cortex, but mainly in the ventricular cavity (the two lateral ventricles forming one cavity). Third ventricle slightly dilated; it held water. Fourth ventricle not dilated. It was found that a membranous septum stretched across the iter, obstructing it completely. There can be no doubt that this also was a case of congenital hydrocephalus, and it seems probable that it was caused by the partition across the iter. This may have been due to intra-uterine inflammation. The mother had had scarlet fever when three months pregnant with this child.

We have now given sufficient proof of our assertion that in almost every case in which posterior-basic meningitis causes hydrocephalus some definite obstruction in the drainage-channel of the brain or cord will be found, if the necropsy be carefully made. We lay stress on this condition; for if the brain be removed in the ordinary way it may be impossible to decide accurately whether the cerebro-spinal foramen be open or closed; and if the spinal cord be neglected an adhesion of the spinal membranes may be overlooked which may account for hydrocephalus with no apparent obstruction within the brain. When the skull-cap has been removed, a wedge-shaped piece of the occipital bone should be cut away extending down to the foramen magnum; the posterior arches of the three uppermost cervical vertebræ must then be cut through, the dura mater carefully divided, and the condition of the subjacent arachnoid accurately noted. Division of this membrane reveals the condition of the posterior subarachnoid space. If then the cerebellum be gently raised from the medulla, it is easy to see whether the cerebro-spinal foramen is open or closed, what its diameter is, and whether there are any morbid adhesions in its neighbourhood. We have employed this method of examination for many years.

The only instances in our experience in which, in hydrocephalus following posterior-basic meningitis, an autopsy made in this manner has failed to discover some organic obstruction, are Nos. 22 and 50. In P.M. 50 we found distinct, though slight, traces of previous meningitis, but no obstruction; yet the whole drainage system of the brain and cord was tensely filled with absolutely transparent fluid. How are we to explain this phenomenon?

It must be remembered that the choroid plexuses derive part of their blood-supply, by means of the "posterior choroid" arteries, from the posterior cerebrals, the terminal branches of the basilar. Inflammation affecting the posterior base of the brain must tend to produce paretic dilatation of the basilar and its branches; the posterior choroid arteries will share in this dilatation, and the choroid plexuses will therefore receive an increased blood-supply. When the inflammatory process implicates the plexuses themselves, their secretion is doubtless diminished, but in the less acute cases it is probably increased. This is probably one factor in the production of hydrocephalus when posterior-basic meningitis causes an obstruction in the drainage-channel, and it may possibly be effective occasionally when no obstruction exists. If the cerebro-spinal fluid be secreted more rapidly than it can escape along the sheaths of the cerebral and spinal nerves, a uniform dilatation of the ventricular cavities and of the subarachnoid spaces at the base of the brain and around the spinal cord, such as we found in case 50, might easily result. When this dilatation had reached a certain amount, it would be aggravated by compression of the membranes of the nerve-roots closing the lymphatic paths in the same way as a pleural effusion closes the lymph-stomata of the pleura.

In P.M. 22 the fact that the lateral ventricles were considerably dilated with clear fluid and the convolutions flattened, while the fourth ventricle appeared to be of normal size, suggested that there had been some obstruction in the iter. This passage, however, was found patent. But some thick yellow lymph was found in the velum interpositum just above the posterior end of the iter; and it seemed to us possible that in this way the iter was compressed, and that thus a real obstruction to the escape of fluid from the lateral ventricles might have existed during life.

The clinical evidence of hydrocephalus consists in prominent fulness of the fontanelle, in separation of the sutures (if these are not already firmly united), in the reappearance of the posterior and lateral fontanelles, and in the increasing size and weight of the head, with tendency to approximate to the spherical form. Sometimes the cranial bones are thinned, and they may even yield on pressure. Occasionally there is slight depression of the eyeballs. All these indications are much less pronounced than in congenital hydrocephalus. If, however, the meningeal inflammation begins in very early infancy, and life is sufficiently prolonged, the hydrocephalus produced may be so extreme as to simulate a congenital case.

We have already referred to the bulging of the anterior fontanelle, which is often observed at the onset of meningitis, and is doubtless due to the initial hyperæmia and the resulting increased outflow of fluid into the lymphatic spaces around the cerebral arteries, all of which open into the general subarachnoid space covering the cortex of the brain. This early bulging of the fontanelle is usually transient.

In some cases of meningitis there is no clinical indication of hydrocephalus, though even in these an autopsy will sometimes prove its existence. In others, after some weeks' illness, a gradual increase in the size of the head is noticed. The fontanelle may be somewhat prominent, or about level, or even slightly depressed; although an autopsy may prove that there is considerable excess of fluid in the ventricles. The increment of size is easily indicated by two measurements—that of the maximum circumference, and that over the vertex from the centre of one auditory meatus to the centre of the other. As a guide to the numbers yielded by these two measurements in healthy children, we offer the following figures, obtained by one of us from the heads of three healthy male infants, weighing respectively at twelve months old 26 lbs. 8 oz., 26 lbs. 2 oz., and 22 lbs. 12 oz. :—

Age.	Maximum circumference.			Over vertex from meatus to meatus.		
2 days	13½	- -	13⅞	9	- -	9
1 week	14	—	—	9	—	—
1 month	14½	15	—	9½	9¾	—
2 months	15¼	—	—	10¼	—	—
3 ,,	16⅛	—	—	10¾	—	—
4 ,,	16½	—	-	11	—	—
5 ,,	16¾	17	—	11	-	—
6 ,,	17¼	—	16¾	11½	- -	11⅜
7 ,,	17½	—	17½	11¾	—	—
8 ,,	17¾	—	17¾	11⅞	—	11½
9 ,,	18	18	—	12	11¾	—
10 ,,	18¼	—	—	12⅜	—	—
11 ,,	—	—	—	—	—	—
12 ,,	18½	—	18¼	12½	—	12¾

We have notes of the presence or absence of indications of hydrocephalus during the course of the illness in 82 cases. In 51 of these there was evidence of hydrocephalus; it was considerable in 16, moderate in 18, slight in 17. In the remaining 31 there was no evidence of hydrocephalus during life, although in some of these it was found after death. The following may be adduced as illustrations.

In a child, aged 6 months, (two months after the onset) the sutures were found to be open. At 8 months the fontanelle was distended, and the lateral sutures open down to a lateral fontanelle on each side, the measurements being

19 and 13½. At 13 months they were 19½ and 14; the lateral sutures were not now felt, but the fontanelle measured 4 finger-tips by 3.

In a second case, at 8 months, the measurements were 18¼ and 11¼; at 9 months, 19 and 11½; at 11 months, 19½ and 12; at 12 months, 19¾ and 12¼. The fontanelle was normal throughout. At 4 years 2 months the figures were 20¾ and 13½, and the child was very backward, being still unable to walk without some support.

In a third child (nearly 4 months old at the onset) there was no indication of hydrocephalus during the first month of his illness. He was not brought again to the hospital until six weeks later, when his mother stated that his head had been enlarging for two weeks. At 6 months old the fontanelle was tense, and 3 finger-breadths wide, and the sutures just open, the measurements being 18 and 11¾. At 7 months old they were 18¼ and 11¾, and the eyes were a little depressed. At 11 months they were 19¾ and 12¼, and the fontanelle was 4 finger-breadths wide.

In a fourth case, in which the illness began at 3 weeks old, at 3 months the fontanelle was distended and the sutures felt. At 5 months old the fontanelle was bulging, and the measurements were 17¾ and 12; at 6 months old, 19 and 13; at 7 months old, 20⅛ and 13¼; at 8 months old, 20¼ and 13½; the fontanelle was 4 finger-tips wide, the head was heavy and fell back. At 2 years 3 months old the figures were 21¼ and 14½.

In a fifth case the illness began at 3 months of age; the head began to enlarge a month later. At 10 months old the measurements were 22 and 16½. At 12 months old they were 23 and 16. The eyes were a little depressed.

In a sixth case (a girl), in which the illness began at 6 weeks of age, the measurements were—at 3 months old, 15½ and 10½ (normal); at 4 months, 16½ and 11; at 6 months, 17⅝ and 11⅝; at 8 months, 19⅜ and 13¼; at 11 months, 21 and 14¾. At 5 months it was observed that the fontanelle was bulging, and the anterior half of the head was rounded. At 7 months lateral fontanelles reappeared. The head was still larger at the time of death, two months later, and the autopsy (P.M. 49) showed enormous distension of the ventricles.

Before concluding this section we wish to offer some suggestions with regard to *the hydrocephalus* found, though usually in smaller amount, *in tuberculous meningitis*, and also with regard to *congenital hydrocephalus*.

In the former condition the same considerations are valid as in the rare cases in which posterior-basic meningitis has caused hydrocephalus, without the presence of organic obstruction. The choroid plexuses receive part of their blood-supply through the "anterior choroid" arteries from the internal carotids, and the vessels composing these plexuses will therefore share in the paretic dilatation caused by the local inflammation at the anterior base. Hence there will be an abnormally rapid secretion of cerebro-spinal fluid.

Again, in tuberculous meningitis there is frequently a considerable deposit of tubercle at the anterior end of the upper surface of the superior vermiform process; and, if the subarachnoid "cistern" immediately in front of this and behind (above) the corpora quadrigemina is filled with inflammatory products, it may possibly exert pressure from above on the posterior end of the iter, which may cause some hindrance to the flow of fluid through this narrow channel. If at the same time the amount of cerebro-spinal fluid secreted be increased, it is easy to understand why the ventricles are found dilated with clear fluid.

With regard to congenital hydrocephalus, we have already shown that some cases are certainly obstructive in origin, and probably due to intra-uterine meningitis. A study of recorded cases of congenital hydrocephalus, such as that of Dr. Ruffer, seems to indicate that a considerable number of such cases are rightly explained by this hypothesis. In many of them the foramen of Majendie was undoubtedly closed, as it was in Dr. Baxter's case, which had previously been under our care at Great Ormond Street. Obstruction of the foramen of Monro is mentioned in other cases; and we have recorded an instance above in which the obstruction was a partition across the iter. Even where no obstruction can be found in the brain, and the cerebro-spinal foramen is widely patent, adhesions of the spinal membranes may have caused an obstruction which is easily overlooked.

There are, however, some cases certainly in which no obstruction can be found, and some in which it cannot exist; as in the cases of hydrocephalus associated with spina bifida, in which puncture of the spina bifida is found to drain the ventricles. We suggest that such are similar to our case No. 50, except in their occurrence at a much earlier period; and that an intra-uterine cerebral inflammation, which had not given rise to organised adhesions, had yet caused such increase in the amount of secretion of cerebro-spinal fluid that it had accumulated more rapidly than it could be removed, and thus the fluid-pressure in the brain had caused congenital hydrocephalus. In some cases, perhaps those in which the lesion occurred at a very early period of fœtal life, the fluid collecting in the spinal canal, and especially in its lower portion, may have been instrumental in producing the spina bifida. Considering the position of the head in the womb, it seems possible that meningocele may be produced in the same way. It is interesting to notice, as Dr. Ruffer points out, that a tumour of this kind always projects through a hole in the bone, and never through a fontanelle; and to compare this fact with the case of hydrocephalus caused by basic meningitis in a syphilitic child which we have recorded above, in which soft swellings, composed of membranes distended with clear fluid, projected through holes in craniotabetic bone; and also with the case recorded by Dr. Baxter.

The thickening of the ependyma of the ventricles, which has been found in cases of congenital hydrocephalus, is probably another result of the inflammation which produced the obstruction to the escape of cerebro-spinal fluid.

Congenital cysts of the brain, containing clear fluid, whether communicating with the ventricular cavities or not, are probably for the most part the result of local softening and subsequent removal of brain-substance, caused by local disease of vessels—thrombosis of arteries or veins; in some cases probably due to syphilis, in others to infective or septic conditions received from the mother.

Pathology.—It can hardly be doubted that in most cases not due to injury meningitis, whether vertical or posterior-basic, is caused by an invasion of micro-organisms. Even where the exciting cause has been a blow or fall, the meningitis may be produced by a growth of microbes permitted by the depression of the normal resistance of the tissues produced by the injury. It is not very uncommon for tuberculous meningitis to follow a blow upon the head (the same thing is true of tumours of the brain in children), and some of these instances are probably not mere coincidences.

A striking fact in the pathology of suppurative vertical meningitis (which usually affects the spinal cord also) is that in a large number of cases it is associated with pneumonia and with other inflammatory visceral lesions. In our 14 cases of this kind (Nos. 1 to 12, 14, and 15) there were only 3 in which there was no visceral lesion beyond slight collapse of the lungs. In 9 of the 14 pneumonia was present; in 5, pleurisy or empyema; in 4, pericarditis; in 1, mediastinitis; in 1, severe bronchitis: in 2 there was lymph on the surface of the spleen; in 2, thrombosis of the cerebral sinuses. In a case under the care of our colleague Dr. Penrose, to which he permits us to refer, a patch of diphtheritic membrane was found in the descending colon, with some small ulcers in its neighbourhood; also a little thin false membrane in the œsophagus.

In one of our cases the meningitis followed erysipelas and cellulitis; and must be looked upon as a septicæmic process. Again, it is possible that the rare cases of meningitis following vaccination, of which we have seen one, may likewise be septicæmic, though it must be admitted that it is sometimes difficult to eliminate the element of coincidence, especially when the vaccination pursues a normal course. The multiplicity of the visceral lesions in vertical meningitis also seems to point to a general infection; and in two of our cases diplococci (apparently M. lanceolatus) were discovered by Dr. Still both in the meningitic exudation and in the inflamed lung or pericardium.

In many cases of vertical meningitis the anterior part of the brain is affected much more than the posterior half, and sometimes almost alone. It is then difficult to avoid the suspicion that the infection may have invaded the subarachnoid space through the lymphatic channels passing through the cribriform plate from the nasal mucous membrane, demonstrated by Key and Retzius. These observers have proved that coloured fluids, injected under low pressure into either the subdural or the subarachnoid space of the spinal cord, make their way into tubular prolongations of the cerebral subarachnoid space surrounding the branches of the olfactory nerves passing through the cribriform plate into the nose. Not only so, but in certain animals they also succeeded in filling, by means of similar injections, a network of lymphatic vessels in the nasal mucous membrane, and furthermore intercellular passages communicating with these vessels, many of which they found to terminate by open mouths on the surface of the mucous membrane. A similar structure has been demonstrated in the bronchial mucous membrane; and it affords an explanation of the fact that the bronchial glands in children are sometimes found to be caseous without any obvious lesion of the bronchi or lungs. If there is thus an open communication between the mucous membrane of the nose and the anterior part of the cerebral subarachnoid space, it is reasonable to suggest that micro-organisms may sometimes enter by this route and thus invade the meninges, even without any obvious lesion of the nose itself.

In the majority of cases of posterior-basic meningitis the path of invasion is probably from the naso-pharynx, through the Eustachian tube and the middle ear, to the membranes of the brain. There are certain anatomical peculiarities in the auditory apparatus of infants which facilitate this invasion. Prof. Macewen points out that the Eustachian tube is shorter, wider, and more horizontal than in the adult; and that the petro-squamosal suture, which passes through the roof of the tympanic cavity, is still incompletely ossified, bringing the membranes of the brain and its subarachnoid space into close relations with the lining membrane of the middle ear. Thus at this early age there is a comparatively open path from the naso-pharynx to the great subarachnoid "cistern" between the cerebellum and the medulla; and also to the smaller, yet relatively large, subarachnoid space above the corpora quadrigemina, in the immediate neighbourhood of the iter, and just in front of the anterior extremity of the superior vermiform process of the cerebellum. The post-mortem evidence given above proves that posterior-basic meningitis usually begins and is most active in one or both of these two sites. It is not often possible to trace any connection between pus in the tympanic cavity and the inflammatory focus in the posterior subarachnoid space; but a similar fact is true in many instances of cerebral abscess resulting from ear disease. In some of our cases there was clear evidence of disease of the middle ear, its mucous membrane being swollen, granular, and injected; and the drum-membrane opaque, thickened, and sometimes perforated. But usually the drum-membrane is entire and the ossicles in place, and the only abnormality is the presence of pus in the tympanic cavity, sometimes also in the mastoid antrum. Too much stress must not be laid upon this—first, because it might possibly be a consequence of meningitis rather than its cause; and, secondly, because the presence of more or less muco-pus in the middle ear is very common in infants who have died from causes unconnected with the brain. Yet two arguments may be adduced in favour of the view that the pus found in the tympanum is in many cases either a main or a contributory cause of the meningitis: the first is that in a large number of cases the symptoms of meningitis definitely follow a catarrh, and that the anatomical relations already mentioned make it easy for the catarrh to spread to the middle ear, and for organisms to be carried thence to the brain; the second is that, in some cases, paracentesis of the tympanic membranes has definitely arrested the disease. It is noteworthy in one case (P.M. 6) that secretion in the tympanic cavities, which seemed no more than "slightly turbid," was found to contain diplococci similar to those present in the meningeal pus and in the inflammatory lymph on the pericardium and the pleura.

Even when the initial catarrhal condition of the middle ear is but slight, it may nevertheless facilitate the ingress of pathogenetic organisms to the brain; just as a catarrhal condition in the fauces, lungs or alimentary canal may facilitate the ingress of the tubercle bacillus.

In the adult the tympanum is completely shut off from the cerebral subarachnoid space, which penetrates into the internal ear but no farther. Key and Retzius, by injection of blue fluids into the subarachnoid space of the spinal cord, were able to obtain partial injection of the peri-lymphatic space of the internal ear, and of the "aquaductus cochleæ," which communicates with it through the "scala tympani" of the cochlea; also of the perineural sheaths of the seventh and eighth nerves in the internal auditory meatus as far as the lamina cribrosa, and in a few cases a little beyond it. But the injection never passed into the endo-lymphatic space or into the "aquaductus vestibuli," which they found to end in a cul-de-sac within the layers of the dura mater, communicating by a fine passage with this space but without communication with the subdural or subarachnoid spaces. And it never passed into the tympanum, proving that in the adult the tympanum is quite separate from the cerebral membranes and the spaces between them. This explains the clinical fact that meningitis and other morbid conditions of the brain resulting from otitis are almost always in adults the consequence of chronic disease of the middle ear, and are very rarely produced by an acute inflammation in previously healthy ears, as is the case in infants.

But there are many cases of posterior-basic meningitis in which the examination of the middle ears shows no morbid condition. Even in these cases we suggest that the micro-organisms may have travelled by this route from the naso-pharynx. Doubtless, if the mucous membrane of the tympanic cavities is diseased, microbes from the pharynx have increased facility of access; but even when the middle ear is healthy the anatomical peculiarities already described appear to make it possible for them to pass from the pharynx to the cerebral membranes. Such microbes are doubtless of many kinds; it is probable that some of them are introduced into the pharynx by breathing air contaminated with sewage emanations.

The condition of the viscera in posterior-basic meningitis shows a most remarkable absence of the inflammatory lesions so frequently found in vertical meningitis. In 5 of our 34 cases of the posterior-basic class (Nos. 13, 16-26, and 29-50) the viscera were not examined, or their condition has not been recorded. Of the remaining 29 the viscera were normal in 21. In 5 there was merely slight collapse of the lungs; in 2 the lungs were congested as well as collapsed; in 1 some tubercle was present in certain organs. But there was not a single case of definite pneumonia, of pleurisy, empyema, or pericarditis. This strongly suggests that these two forms of meningitis are really different diseases—that they are due to different organisms. It may be added that while vertical meningitis is usually highly suppurative and very extensive, posterior-basic meningitis is much more local and much less suppurative—the former more acute and more rapidly fatal, the latter chronic and tending to form organised adhesions. These differences increase the probability that the two forms of meningitis are the results of the growth of microbes of diverse nature. The question must be settled by bacteriology: it is therefore desirable to state the observations which have already been made with regard to this question.

Several bacteriologists have found the diplococcus of Fränkel (D. pneumoniæ, M. lanceolatus, Pneumococcus) in the purulent exudation of cerebro-spinal meningitis. "Thus Netter (1889), in a summary of the results of researches made by him in 25 cases of purulent meningitis, reports as follows:—Thirteen cases were examined microscopically, by cultures, and by inoculations into susceptible animals; 6 cases by microscopical examination and experiments on animals; and the remainder only by microscopical examination. Four of the cases were complicated with purulent otitis, 6 with pneumonia, 3 with ulcerative endocarditis. The pneumococcus was found in 16 of the 25 cases; in 4 S. pyogenes was present; in 2 D. intracellularis meningitidis of Weichselbaum; in 1 Friedländer's bacillus; in 1 Neumann and Schäffer's motile bacillus; in 1 a small curved bacillus. In 45 cases, collected from the records of the subject by Netter, this micrococcus was present in 27, Streptococcus pyogenes in 6, and D. intracellularis meningitidis of Weichselbaum in 10. Monti (1889) in 4 cases of cerebro-spinal meningitis demonstrated the presence of the same micrococcus. In 3 of his cases pneumonia was present also. In 2, Staphylococcus pyogenes aureus was associated with D. pneumoniæ" (Sternberg). "In otitis media this micrococcus has been found in a considerable number of cases in the pus obtained by paracentesis of the tympanic membrane, and quite frequently in pure cultures: by Zaufal (1889) in 6 cases; by Levy and Schrader (1889) in 3 out of 10 cases in which paracentesis was performed; by Netter (1889) in 5 out of 18 cases occurring in children" (Sternberg). It has also been found by various observers in ulcerative endocarditis and in acute abscesses and in suppurative arthritis occurring as complications of pneumonia.

Weichselbaum, in 6 cases of cerebro-spinal meningitis, found and cultivated a diplococcus not previously known; he observed it usually in the interior of the pus-cells, and therefore called it "D. intracellularis meningitidis." It differs from Fränkel's diplococcus in not being stained by Gram's method, and in some peculiarities of growth in artificial culture; but resembles it in its brief period of life in such cultures, which is limited to a few days. Weichselbaum's diplococcus is highly pathogenetic when injected into animals: mice die within two days after inoculation; injections into the meninges of dogs produce a purulent hæmorrhagic meningitis. This microbe has been found by many subsequent observers in cases of epidemic cerebro-spinal meningitis, both after death and in the fluid withdrawn from the spinal subarachnoid space by puncture of the membranes in the lumbar region of the spine.

callosum caused a similar degeneration to each of these experiments alone, but more extensive;—a fact which he explains on the supposition that half of the fibres of the corpus callosum have the left hemisphere as their trophic centre, and the other half the right hemisphere. Hence, on cortical lesion of one hemisphere the fibres from that hemisphere degenerate; while on section of the corpus callosum the fibres of the opposite hemisphere also degenerate, and thus cause a double degeneration of the callosal fibres on the side of cortical lesion. The experiments of Turner and myself on removal of the occipital lobe and division of the corpus callosum confirm as regards the visual centres those of Muratoff on the motor centres.

Mott and Schäfer (9) found that on stimulation of the uncut surface of the corpus callosum movements were caused on both sides of the body; while on irritation of the cut surface movements were obtained only on the side opposite that of irritation. From these experiments they conclude that the movements depend on indirect excitation of the motor centres through the callosal fibres,—a conclusion which was confirmed by the fact that the movements did not occur if the motor centres were extirpated. It would appear, therefore, from these experiments, that the corpus callosum must contain commissural fibres connecting the motor centres with each other; but, inasmuch as the movements were not in all cases strictly localised, they are of opinion that the fibres, though mainly massed in definite parts of the corpus callosum, are not entirely confined to those parts, but are, to a certain extent, scattered along the middle thinner part of the commissure.

I have found that section of the corpus callosum in monkeys does not cause any obvious symptoms, sensory or motor; and Koranyi, from similar experiments on the corpus callosum of dogs, has arrived at the same conclusion.

In man pure uncomplicated destructive lesions of the corpus callosum are practically unknown. A few cases of congenital absence or defect of the corpus callosum have been described, but, as in them the brain was otherwise malformed, no conclusions concerning the functions of the corpus callosum can be founded upon them.

Tumour is the chief form of disease affecting the corpus callosum, while vascular lesions are exceedingly rare. Erb and Hougberg have, however, each recorded a case of complete, or almost complete, destruction of the corpus callosum by hæmorrhage; and Kaufmann has described a case of complete destruction of the corpus callosum by embolic softening, secondary to aneurysm with meningitis. The last had been very imperfectly observed during life, but practically no symptoms referable to the corpus callosum were found in any of the three cases. Erb concludes, in accordance with my own and Koranyi's experimental results, that in an adult with no previous brain disease, almost the entire corpus callosum can be destroyed without any disturbance of motion or co-ordination, or of sensation, general or special; and without any noteworthy disturbance of intelligence.

Tumours of the corpus callosum almost always invade the hemispheres on one or both sides, and the probability is that the symptoms which have been observed were really due to local damage, or to general pressure on the hemispheres.

Bristowe, however, was of opinion that the symptoms which he observed in four cases are so characteristic as to enable one to arrive at a fairly accurate regional diagnosis. These are:—

1. An ingravescent character.
2. The gradual onset of hemiplegia.
3. The association of hemiplegia on the one side with vague hemiplegic symptoms on the other.
4. The supervention of stupidity, generally with extreme drowsiness; a puzzled, inquiring look; difficulty of swallowing, and speechlessness, due mainly to incessant drowsiness and apathy without any actual paralysis.
5. Absence of direct implication of the cranial nerves.
6. Death from coma.

There is also, according to Bristowe, absence, or insignificance, of the more striking symptoms of cerebral tumour. Headache is never very severe; there is practically no sickness, and there are no convulsions. Optic neuritis was entirely absent in one case.

Giese agrees with Bristowe in all essential points, and further adds that if symptoms occur pointing to the corpus callosum as the seat of the disease, and there are also cerebellar symptoms, then it is highly probable that the tumour has its seat in the splenium of the corpus callosum.

Bristowe's conclusions have also been supported by Bruns.

On examination of sixteen recorded cases I find disturbance of intelligence, chiefly in the form of progressive mental dulness or dementia, more or less in all; optic neuritis was present in nine—three of which, however, were doubtful; headache in seven; hemiparesis in six; paraparesis in four; vomiting in three.

Summary.—While, in the present state of our knowledge, we may say that there are no symptoms due to damage of the corpus callosum as such, still the presence of such a combination of symptoms as that enumerated by Bristowe enables us to make a fairly accurate, though never more than a probable diagnosis of tumours implicating the corpus callosum.

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Lesions of the Centrum Ovale and Internal Capsule.—The centrum ovale, or white substance of the cerebral hemispheres, contains not only projection fibres, which by means of the internal capsule connect the cortex cerebri with the periphery, but also fibres which connect the cortex with the optic thalamus, and, according to some views, but less certainly, with the corpus striatum also. Besides this mass of fibres, which constitutes the corona radiata, the centrum ovale contains several systems of fibres, some of which are intra-hemispherical, or associating and connecting different parts of the hemisphere with each other; while others are inter-hemispherical or commissural, such as the corpus callosum and anterior commissure, which probably connect corresponding parts of the hemispheres with each other.

As to the functions of the various systems of fibres, which together constitute the centrum ovale, we know nothing with certainty beyond those which form the internal capsule; nor is it always possible to distinguish between lesions of the cortex itself and the subjacent fibres of the corresponding lobes or convolutions. It is desirable for topographical purposes to indicate the position of lesions of the centrum ovale with reference to definite regions of the cortex. For this purpose Pitres has suggested a convenient method of dividing the hemisphere into a series of sections parallel to the fissure of Rolando, or vertical to the long axis of the hemisphere.

The first section through the prefrontal area forms the *prefrontal* section.

The second section through the base of the frontal convolutions forms the *pediculo-frontal* section, which is divided into a superior, middle, and inferior fasciculus corresponding with the respective frontal convolutions.

The third section through the ascending frontal or precentral convolution forms the *frontal* section, which is also divided into a superior, middle, and inferior fasciculus.

The fourth or *parietal* section is formed in a similar manner by section through the ascending parietal or post-central convolution. This also is divided into a superior, middle, and inferior fasciculus.

The fifth is formed by dividing the hemisphere three centimetres posterior to the fissure of Rolando, and, passing through the superior and inferior parietal lobules, constitutes the *pediculo-parietal* section, which is composed of a superior and inferior pediculo-parietal and *sphenoidal* fasciculus.

The sixth is formed by similar division through the occipital lobe, and constitutes the *occipital* section, in which no separate fasciculi are differentiated.

What has been said above with respect to the symptomatology of the prefrontal, occipito-angular, and temporo-sphenoidal lobes is essentially true of lesions of the prefrontal, pediculo-parietal, and occipital sections of the centrum ovale. It is only lesions of the medullary fasciculi which correspond to the fronto-parietal area which cause paralysis of motion and degeneration of the motor tracts of the internal capsule. Such lesions cause symptoms in most respects like those of lesions of the cortex itself in the corresponding regions; that is to say, they cause complete hemiplegia, similar to that resulting from total destruction of the Rolandic area, or of the motor division of the internal capsule; or monoplegia, according as the lesion is limited to the tracts corresponding with the respective cortical centres. Many cases of this kind have been recorded by Pitres, and a large number of those which have been referred to in the previous pages have involved the medullary fibres of this region as well as the cortex itself.

There are few cases on record of monoplegias or dissociated paralyses due to limited lesions of the centrum ovale, not also invading the cortex. Most of such lesions have been situated at the foot of the corona radiata, just where the projection fibres enter the internal capsule. An interesting case of this kind has been reported by Bennett and Campbell. This was a case of complete paralysis of the left arm, with unimpaired sensibility, which after death was found to be due to a circumscribed softening of the centrum ovale at the point where the medullary fibres of the middle parietal fasciculus converge into the internal capsule, just posterior to the knee. Cases have been reported, by various authors, which show that lesions limited to the lower fasciculus of the frontal section,—that is, the medullary fibres subjacent to Broca's convolution, —may cause aphasia without any obvious affection of the cortex itself.

It is probable, however, that in these cases the lesion is such as to impair the nutrition, and therefore the functions of the cortex itself; or it may be that it destroys the associating fibres which connect the speech centre with those of sensory perception and registration.

While symptoms of destructive lesion of the centrum ovale of the fronto-parietal or Rolandic area cannot be easily distinguished from those affecting the cortex itself, it is otherwise when the lesions are of an irritative character. Irritative lesions of the motor cortex are, as we have seen, characterised by the occurrence of tonic, followed by clonic spasms of an epileptiform character. Irritative lesions of the medullary fibres of the Rolandic area cause only tonic, and not the repeated, or clonic spasms of the cortical type. It has been proved experimentally that electrical irritation of these fibres causes only tonic contraction proportional in duration to the duration of the stimulus; and there is reason for believing, as Pitres has shown, that the early rigidity which is seen in some cases of cerebral hæmorrhage, and which has been generally attributed to the inundation of the ventricles, is in reality due to irritation of the fronto-parietal fibres of the centrum ovale in the course of their convergence into the internal capsule.

If, however, the lesions are immediately subjacent to the cortex, and are such as to cause irritation of the cortical gray matter also, convulsions of the usual epileptiform type may ensue. This occurs not infrequently in hæmorrhage of the centrum ovale; and the occurrence of convulsions of the Jacksonian type in cases obviously hæmorrhagic in character has been considered by some to be diagnostic of hæmorrhage immediately subjacent to the cortex. (Von Bamberger.)

Of the symptomatology of lesions of the anterior, or *lenticulo-caudate*, division of the internal capsule we know little with certainty. The fibres of this portion radiate into the prefrontal and post-frontal lobes, and they degenerate downwards towards the pons when those regions are the seat of destructive lesions. Their position in the foot of the crus cerebri and probable distribution will be described in connection with the lesions of the crura themselves (p. 343).

Horsley and Beevor have shown that by electrical irritation of the internal capsule of the monkey, movements of the head and eyes were caused on irritation of the posterior part of the lenticulo-caudate segment, just anterior to the knee. It would appear, therefore, that this region of the internal capsule corresponds to the cortical centres of the head and eyes, situated, as we have seen, in the post-frontal area.

The posterior, or *lenticulo-optic*, segment of the internal capsule forms with the anterior segment an acute angle termed the *knee*, as seen in horizontal sections. The anterior two-thirds of this segment, including the knee, contain the pyramidal fibres of the motor cortex; and, according to the experiments of Horsley and Beevor, the fibres are so arranged that those from the inferior cortical centres are situated most anteriorly, while those from the superior are situated most posteriorly. Hence irritation from the knee backwards causes, in order, movements of the tongue, mouth, upper limb (shoulder preceding the thumb), trunk, and lastly lower limb (hip preceding the toes).

It is theoretically possible that lesions of the internal capsule may be so limited as to cause monoplegia of the cortical type, but this must be exceptional, as the pyramidal fibres of the different cortical centres are so closely congregated together that a focus even of small magnitude must necessarily involve several tracts. The case reported by Campbell and Bennett (*supra cit.*) was of this exceptional character, and the position of the lesion at the point where the coronal fibres converged into the internal capsule corresponded very closely with the position of the arm fibres in Horsley and Beevor's scheme. That the tracts for the mouth and tongue are situated in the knee of the internal capsule is verified by the occurrence of secondary degeneration in this region in cases of long-standing aphasia, such as have been reported by Charcot and Brissaud, Ross, and others. This tract has been termed by Charcot and Brissaud "the geniculate fasciculus," and the fibres from this region are continued downwards in the mesial portion of the crus cerebri. The position of the pyramidal fibres for the leg at the junction of the posterior with the anterior two-thirds of the lenticulo-optic segment is supported by the fact that in hemiplegia, due to lesions of the internal capsule, in which sensation is notably affected, the leg is frequently more severely and more enduringly paralysed than the arm; thus reversing the order of events in ordinary hemiplegia.

The posterior third of the lenticulo-optic or hinder segment of the internal capsule constitutes, to use Charcot's name for it, the "carrefour sensitif," or sensory crossway, by which the impressions of general sensibility ascend to the cortical centres. With these appear to be associated also the tracts of special sensation, namely, hearing, taste, and smell, together with the optic radiations, situated most posteriorly, which ascend to the occipito-angular region. There is still some doubt as to the path by which olfactory impressions are conveyed to the opposite hemisphere, as we have already seen that the anatomical facts are more in favour of a direct connection of each olfactory tract with the hemisphere on the same side, and a decussation in the anterior commissure is far from clearly established. It is to injury of this portion of the internal capsule, in all probability, that the hemianæsthesia is due which occurs in connection with lesions due to blocking or rupture of the lenticulo-optic artery, and in connection with diseases affecting the optic thalamus. The hemianæsthesia does not necessarily affect all forms of sensibility alike. The most common form is hemianæsthesia of tactile and general sensibility, as well as of the so-called muscular sense. In many cases there is also homonymous hemianopsy, owing to implication of the optic radiations; and that hemianopsy is in the vast majority of instances due to lesion in this region is proved by its common association with hemianæsthesia and a greater or less degree of hemiplegia.

Hemianæsthesia is frequently of a functional character, as has been pointed out by Charcot and his pupils. In this condition there is loss not only of general and muscular sensibility, but also loss or diminution of hearing, taste, and smell on the opposite side, as well as profound impairment of vision in the opposite eye, characterised by diminution of visual acuity, and concentric contraction of the visual field with dyschromatopsy. The contraction of the visual field occurs to some extent also in the eye on the same side. The visual defect in this form of hemianæsthesia is not of the homonymous hemiopic, but of the cross-amblyopic form.

That conditions similar to those of hysterical hemianæsthesia may occur in connection with actually demonstrated or highly probable organic lesions of the internal capsule is evidenced by cases reported by Féré, Gowers, and myself already referred to (p. 314).

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LESIONS OF THE CORPORA STRIATA.—The special functional relations of the corpora striata are still doubtful. They cannot be exposed for experimental purposes without inflicting a certain amount of damage on other centres. When they are exposed in monkeys and other animals by division of the corpus callosum, I have found that electrical irritation of the free nucleus caudatus gives rise to contraction of a tonic character of all the muscles on the other side of the body, except in rabbits, in which clonic movements of the jaws occur during continuance of the stimulation. This general pleurotonic spasm instantly ceases when the electrodes are transferred to the surface of the optic thalamus. From these phenomena I have argued that the corpora striata are related to the movements of the opposite side of the body.

Frank and Pitres and others are of opinion that the movements which I have described are not the direct result of irritation of the gray matter of the corpus striatum, but are due to the diffusion of the irritation to the adjacent motor tracts of the internal capsule. But this explanation does not appear to me consistent with the fact that similar irritation of the optic thalamus, which is quite as close to the motor tracts of the internal capsule, is entirely negative.

Nothnagel found a certain spot in the nucleus caudatus, situated close to the third ventricle, to be mechanically irritable. He states that puncture of this spot in rabbits with a fine needle, or injection therein of a few drops of chromic acid, causes an irresistible tendency on the part of the animal to run or jump until it becomes exhausted. The fact that the internal capsule is undoubtedly excitable by electrical stimulation does not, therefore, exclude the possibility of a direct irritability of the gray matter of the corpus striatum any more than that excitability of the corona radiata excludes the direct excitability of the motor cortex.

Destructive lesions of the nucleus caudatus and nucleus lenticularis cause, according to Nothnagel's experiments, motor disturbances of the opposite side, but more marked in the case of the lenticular than of the caudate nucleus. And Carville and Duret found that destruction of the ganglia of the corpus striatum, together with the tracts of the internal capsule which lie between them, causes a much more complete hemiplegia than that due to the lesions of the motor cortex or pyramidal tracts. Experimental evidence, therefore, is in favour of the view that the ganglia of the corpus striatum have some relation to the movements of the opposite side of the body, even though the exact nature of this relation is not clearly indicated.

The facts of clinical observation in man are somewhat conflicting. The corpora striata, with adjacent tracts, are frequently the seat of destructive lesion, of which the most common is hæmorrhage. The favourite seat of cerebral hæmorrhage is from rupture of the lenticulo-striate branches of the middle cerebral artery. This is apt to destroy the gray matter both of the caudate and lenticular nucleus, as well as the tracts of the internal capsule. Less frequently the hæmorrhage occurs from rupture of the lenticulo-optic arteries which supply the lenticular nucleus and anterior part of the optic thalamus. Hæmorrhage in this region is especially apt to injure the posterior or sensory tracts of the internal capsule. More rarely it occurs in the anterior part of the caudate nucleus from rupture of some of the branches of the anterior cerebral artery. Hæmorrhagic lesions of the corpus striatum, in the first instance at any rate, invariably produce hemiplegia of motion alone, or of motion and sensation; the latter more particularly when the rupture occurs in the lenticulo-optic artery. Inasmuch, however, as hæmorrhagic lesions are sudden, and affect directly or indirectly not only the gray matter of the ganglia, but also the tracts of the internal capsule, it is impossible to distinguish hemiplegia so caused from that occasioned by lesion of the internal capsule itself. An examination, however, of the facts of stationary lesions, such as softening confined to the gray matter of the ganglia, and cysts due to antecedent hæmorrhage, has shown that extensive lesions of this character may be found in the ganglia of the corpus striatum without any symptoms of paralysis, motor or sensory; and the same is true of tumours.

Many examples might be cited from clinical records, which it would be impossible to refer to in detail. But we may take it as a well-established fact that stationary lesions of variable size may be found in the gray matter of the corpus striatum, either in the nucleus caudatus or nucleus lenticularis, without the manifestation of any symptoms of motor paralysis during life.

Pseudo-bulbar paralysis.—In relation, however, with affections of the corpus striatum, it will be convenient to discuss in somewhat greater detail the pathology of pseudo-bulbar paralysis, which by several observers has been attributed to lesions of the lenticular nucleus. I have already (p. 296) described a group of symptoms simulating bulbar paralysis, resulting from bilateral lesion of the cortical centres of articulation. A similar group of symptoms has also been frequently observed associated with lesions, more especially bilateral, affecting the region of the lenticular nucleus and motor segment of the internal capsule. Generally there are two distinct attacks of hemiplegia, often separated by a long interval; and it is only after the second attack, as a rule, that the characteristic bulbar symptoms become manifested. In this condition there is generally a dull, apathetic expression and impaired articulation, the speech being slow, indistinct, monotonous, and with nasal intonation. There is paresis or paralysis of the lips, tongue, and palate, generally also some difficulty in swallowing. Though the vocal cords are, as a rule, unaffected, yet a few cases have been reported in which paresis or paralysis of one or both vocal cords has been noted (Oppenheim and Siemerling, and Münzer).

Of great importance as a means of distinction between pseudo-bulbar and true bulbar paralysis is the absence in the paralysed parts of atrophy, fibrillary tremors, alteration in the electrical contractility of the affected muscles, or affection of sensation. The reflexes of the palate and pharynx are as a rule unimpaired, but they have occasionally been found diminished or even lost. (See cases reported by Lepine, Ross, Colman, Oppenheim and Siemerling, Hughlings Jackson.)

The limbs are generally more affected on one side than the other (the legs more than the arms); and there is often a tendency to convulsive laughing or crying, especially on attempts to speak. Respiratory and circulatory disturbances are as a rule absent, though they have been observed in some cases. In most of the recorded cases the lesions, generally hæmorrhagic, or foci of softening, have been bilateral, and more or less symmetrical in the two hemispheres. In a few instances, however, the lesion has been found in one hemisphere only. (See cases recorded by Magnus, Kirchhoff, Ross, Drummond, Bamberger, Wharton Sinkler.)

The lesions have been found in the cortex, in the subcortical white matter, or in the central ganglia. The most common seat of the lesion is the basal ganglia and neighbouring white substance. As a rule the lesions are more or less extensive and diffuse, implicating the central ganglia as well as the capsular fibres; at other times they may be circumscribed, and then they are usually in the lenticular nucleus in its outer segment or putamen. But in these cases it is difficult to eliminate possible implication of the internal capsule, especially in the region of the knee. Cases, however, in which the lesions appear to have been strictly confined to the external division of the lenticular nucleus have been recorded by Lepine and Leresche. Eisenlohr's case is one in which the lesion was confined to the optic thalamus and caudate nucleus, without affection of the lenticular nucleus (6). Though the pons and medulla have been stated to have been entirely free from lesion in many recorded cases, yet from the degree of paralysis of the palate, pharynx, and vocal cords, which have been reported in some of then, one cannot help suspecting, in accordance with the views of Oppenheim and Siemerling, that there have been undetected lesions in this region. These authors, from an examination of five cases observed by themselves, as well as a review of the literature on the subject, have arrived at the conclusion that in the majority of carefully examined instances there are found, in addition to lesions in the hemispheres and ganglia, also small foci of softening in the medulla and pons. These were present in all the five cases examined by them. There is no doubt that in a large number of cases of pseudo-bulbar paralysis—in more than half, according to Boulay—there is sclerosis of the basilar vessels, thus favouring the occurrence of hæmorrhage, or softening of a multiple character, not only in the central ganglia, but also in the pons and medulla. The occurrence of symptoms of pseudo-bulbar paralysis from bilateral lesion of the centres and conducting tracts of articulation is readily understood, but it is more difficult to give a satisfactory explanation of the cases of unilateral lesion, as the muscles of articulation and deglutition are innervated from both hemispheres. It is possible that in these cases the centres of one side may predominate; but this is unlikely, and it is more probable that there have been undetected lesions on the other side. In those cases in which the lesions have been apparently limited to the lenticular nucleus, which has been by some regarded as an independent centre for the movements of articulation and deglutition, it is difficult to harmonise the symptoms with the negative results of similar lesions; and the probability is that, even where not demonstrated, there have been lesions also of the internal capsule, if we assume that they have been entirely absent from the pons medulla.

In connection with lesions of the lenticular nuclei, we may here refer to the symptoms met with in three members of one family, reported by Homén. The symptoms were giddiness, a drunken gait, indistinct speech, impairment of memory and gradually increasing dementia, with rigidity of the whole body, especially of the legs, which became fixed in the flexed position. The necropsy in two of the cases revealed softening in each lenticular nucleus. Some microscopical changes were, however, also demonstrated in the cells and fibres of the cortex.

Certain facts of experiment, as well as of clinical observation, have been quoted as showing a relation between the corpus striatum and the temperature of the body. Aronsohn and Sachs found that mechanical and electrical irritation of the caudate nucleus, near the so-called "*nodus cursorius*" of Nothnagel, caused a notable elevation of temperature, with sensible increase of the respiratory energy and excretion of urea. Dr. Hale White (20) has found that puncture of the corpus striatum in rabbits causes a temporary rise in temperature, the highest rise being when the puncture was made anteriorly, averaging in this case about one degree Fahrenheit. To produce this effect it was necessary that the lesion should be bilateral. Ott, however, has found that in birds puncture of the corpora striata generally caused a fall of temperature, except when the hinder part was injured, in which case it rose about 1° F. The experimental evidence is thus conflicting.

Several clinical cases have been quoted by Hale White in support of his experimental results. Cases have also been reported by Nothnagel, Kroemer, and others, in which marked vaso-motor disturbances on the opposite side of the body were associated with lesions in the corpus striatum. Thus Kaiser reports a case of vaso-motor paralysis of the right limbs with a temperature in the right axilla varying from 1° C. to 15° C. higher than in the left, associated with softening in the left gyrus supramarginalis, the left caudate nucleus, and a small cyst in the left lenticular nucleus. The lesion in the supramarginal gyrus was old, and Kaiser attributes the rise of temperature and the vaso-motor paralysis to the lesions in the corpus striatum, especially of the caudate nucleus, which were more recent; and the œdema was only observed about two months before death. This case confirms a similar one reported by Guicciardi and Petrazzoni, in which a small hæmorrhagic cyst in the left caudate nucleus was associated with right hemiplegia, hyperæmia and hyperthermia.

Beyond quoting these facts and opinions, however, it does not seem possible, in the present state of our knowledge, to express any definite opinion on the relation of the corpus striatum to thermal and vaso-motor innervation.

Summary.—(i.) Lesions of the lenticular and caudate nuclei may be entirely latent.

(ii.) Lesions, such as hæmorrhage, involving the internal capsule as well as the nuclei of the corpus striatum, cause hemiplegia of the opposite side, with or without affection of sensation, according as the lesion invades both the anterior and posterior division of the lenticulo-optic segment of the internal capsule.

(iii.) Pseudo-bulbar paralysis results from bilateral lesions in the ganglia of the corpus striatum and related fibres of the internal capsule.

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Lesions of the optic thalami.—What has been said of the obscurity of the functional relations of the corpora striata is true also of the optic thalami. It is difficult to establish experimental lesions in these ganglia without inflicting injury on other parts, and clinical cases occur very rarely in which the lesion is limited to the optic thalamus, or in which an indirect effect on neighbouring structures may not be assumed as possible. But, while this is true, there are certain symptoms which, as clinical experience teaches us, point with considerable probability to a lesion implicating the optic thalamus, although we may not be able to discriminate between the direct and indirect effects of the lesion. The forms of lesion which are most common in the optic thalamus are:—

(i.) Hæmorrhage; (ii.) softening; (iii.) tumours.

(i.) *Hæmorrhage.*—As has already been stated, the central ganglia are the most frequent seat of cerebral hæmorrhage, and the rupture often extends into the optic thalamus, but does not often begin in it. Andral found that in 301 cases of cerebral hæmorrhage affecting the corpus striatum and optic thalamus, only 35 were confined to the optic thalamus itself. In rupture of the lenticulo-optic arteries, the extravasation begins in the thalamus, or between it and the corpus striatum; and it often damages the posterior part of the internal capsule. The small vessels which proceed from the posterior cerebral or posterior communicating arteries to the inner portion of the optic thalamus usually cause hæmorrhages near the surface, which are prone to rupture into the ventricle. Those from the posterior cerebral, which supply the hinder part of the optic thalamus, may give rise to hæmorrhage into the posterior part of this ganglion, which extends either into the ventricle or downward into the crus or pons.

(ii.) *Softening* in the optic thalamus, as in the corpus striatum, is usually the result of arterial sclerosis, or syphilitic endarteritis.

(iii.) *Tumour* of the optic thalamus is relatively infrequent; and still more rarely is it limited to the optic thalamus.

Affections of motion.—Hemiparesis or hemiplegia is generally present in lesions implicating the optic thalamus. Thus we find it in all of thirteen cases of tumour, in all of four cases of hæmorrhage, and in five of six cases of softening. But there is reason to believe that the motor symptoms are not due to lesion of the optic thalamus as such, but to direct or indirect implication of the internal capsule or cerebral peduncle. When paralysis is found associated with old thalamic foci, we find them usually in the middle third or centre of the ganglion. This is in harmony with the view that the paralysis is in reality due to implication of the internal capsule, as lesions in this situation are more likely to affect the motor tracts. These views are fairly in accordance with the results of experiment. Electrical irritation of the part has always failed in my hands to produce muscular contraction on the opposite side of the body. Nothnagel has not observed motor paralysis after lesions of the optic thalami. Dr. Turner and I, in a recent experiment on a monkey, found that almost total destruction of the optic thalamus caused weakness on the opposite side of the body, which disappeared within a week; and the probability is that the hemiparesis in this case was due to indirect implication of the internal capsule.

When paralysis occurs in man, as the result of lesion of the optic thalamus, the clinical picture does not appear to be quite so uniform as that dependent on lesion of the corpus striatum. The face and tongue are less frequently affected than in the latter case, and sometimes isolated paralysis of the arm has been observed. In a case of tumour reported by Lloyd the arm was totally paralysed, the leg paretic, and the face normal; in one by Fisher the arm and face were early affected, and the leg at a later date. On the other hand, Eisenlohr (5) records a case of symmetrical softening of the pulvinar, on both sides, in which the legs were specially weak, the arms slightly so, and the face normal.

A curious combination of symptoms has been observed by Meynert in two cases of lesion of the optic thalamus; namely, flexion of the arm opposite and extension of the arm on the side of lesion, the head in both cases being turned towards the opposite side. Schiff and Nothnagel have described similar symptoms after experimental lesions of the optic thalamus. Michell Clarke, however, has recorded a case of glioma affecting the left optic thalamus and pressing on the internal capsule, which caused right hemiparesis with flexion of the right arm and extension of the right leg, the head and eyes being turned at first to the left, but later to the right. This case differs from those of Meynert in that the arm on the side of lesion was not extended. The motor weakness observed in connection with lesions implicating the optic thalamus is seldom absolute, and often is a mere paresis, or paresis with rigidity.

In a case of soft vascular tumour under my own care there appeared to be hemiparesis; but on testing the movements of the different joints I found a stiffness or rigidity rather than actual loss of power.

Relation to mimetic movements.—Nothnagel states that if, in a focal lesion with hemiplegia and facial paralysis, voluntary movement of the facial muscles is abolished, while the two sides of the face are alike in the expression of emotion, such as laughing, crying, pain, etc., we may assume that the optic thalamus and tracts connecting it with the cortex are intact.

Many cases have been reported which corroborate this view. Thus Zenner reports a case of lesion of the optic thalamus in which paresis of the face was more marked in laughing than when showing teeth voluntarily. I have observed the same in a case under my own care; and Kirilzew has reported a case in which, though the voluntary movement of the face was equal on both sides, the right was quite motionless in laughing or screaming. On the other hand, cases have been reported in which with lesion of the optic thalamus there has been no defect in the mimetic movements. Thus Eisenlohr (5) has reported the case of a cavity occupying the greater part of the left optic thalamus and posterior part of the internal capsule, in which there was no permanent affection of the mimetic action of the face. A similar case has been reported by Jakob; and Senator has described a case of tubercle invading the whole of the left optic thalamus, in which there was slight obliteration of the right naso-labial fold, which, however, disappeared on crying. It would appear, therefore, on consideration of these somewhat contradictory facts, that the relation of the optic thalamus to mimetic movements has not yet been satisfactorily established.

Bechterew, from experiments on rabbits, finds that irritation of the optic thalami sets in action a complex of movements necessary for the nutrition of the organism, beginning with those of mastication and deglutition, and ending with those of the bowel. And he formulates the view that the optic thalami are the centres for the various vegetative functions of the organism, and of the involuntary movements expressive of feeling or emotion. In reference to this hypothesis it is enough to say that little or nothing beyond the doubtful relation of the optic thalamus to psycho-reflex mimetic movements has been established by clinical observation.

Post-hemiplegic chorea.—Among motor disorders met with in connection with lesions implicating the optic thalamus and neighbourhood, unsteady or chorea-like movements in the affected limbs have frequently been observed, more particularly in those cases in which there has been considerable recovery of volitional control. These movements may occur, to some extent, spontaneously; but they are especially marked on volitional exertion. They are allied to the mobile spasm of athetosis, and the condition is generally named "hemiplegic chorea." Hemiplegic chorea is usually, in the earlier stages of the affection at least, accompanied by some degree of hemianæsthesia. The seat of the lesion has been, for the most part, in or outside the optic thalamus, implicating, it may be, the caudate or lenticular nucleus also; and in many instances it has actually involved the internal capsule, more particularly its posterior or sensory division. It is doubtful how far we should ascribe the choreic movement to lesion of the optic thalamus, as such; or to the internal capsule. The latter is the more probable view, and the phenomena are perhaps occasioned by some irritation set up by the lesion. Often, in connection with tumour of the optic thalamus, the opposite limbs, more particularly the arm, are affected with incoördination varying from slight instability to pronounced ataxy, and frequently exhibit the characters of disseminated sclerosis. The choreic, unsteady, and ataxic movements are all probably but varying degrees of the same affection. Unstable or choreic movements in hemiparetic limbs, however, are not necessarily indicative of lesion in the optic thalamus, or external to it; as they may occur also in connection with lesion of the cortex, crus, pons, and other parts.

The pathology of these choreic and ataxic disorders has been the subject of considerable speculation, but it cannot be said that anything satisfactory has been as yet established.

Sir J. Crichton-Browne is of opinion that considerable destruction of the optic thalamus causes diminution or loss of reflex excitability, but this is by no means a constant symptom, and where it has been observed the lesion has not been limited to the thalamus itself. The state of the knee-jerk varies. In some cases it has been found defective or absent, as in a case of tumour of the left optic thalamus reported by Lloyd. But again, as in a case reported by Eisenlohr (5), of symmetrical softening of both pulvinars, both knee-jerks were increased, and double ankle clonus set in. Hence, neither as regards the superficial nor the so-called deep reflexes can any definite relation to the optic thalamus be considered as established.

Affections of sensation.—Hemianæsthesia, or impairment of sensation, has been frequently observed in connection with lesions implicating the optic thalamus. Thus, of four recorded cases of hæmorrhage which I have examined, there was hemianæsthesia as well as hemiplegia in all. Of thirteen cases of tumour, five had hemianæsthesia, and two analgesia also. One had anæsthesia of the face only (Sinkler); one had anæsthesia of the leg only (Michell Clarke); one had anæsthesia and analgesia of the arm (Fisher); and in five there was no affection of sensation. Of six cases of softening, in one sensation was unaffected (Zacher); one had almost total hemianalgesia (Eisenlohr, 5); one had severe paræsthesia (Edinger); while in the others the condition of sensation was not specially mentioned. The cases, however, are so numerous in which sensation has been unaffected, notwithstanding the existence of lesions in the optic thalamus, that we cannot attribute the loss of sensation in these in which it has been observed to lesion of the optic thalamus itself; and the probability is that the symptoms have been actually due to direct or indirect implication of the posterior or sensory tracts of the internal capsule. Tingling, and other forms of paræsthesia, are not infrequent in cases in which there is no obvious loss of sensation; and in some cases there is a painful hyperæsthesia conditioned without doubt by irritation of the sensory tracts of the internal capsule. Thus Edinger records a case of softening in the left optic thalamus in which the focus lay directly on the sensory fibres of the internal capsule. On the day after the onset there was right hyperæsthesia, followed by violent pains in the right half of the body, pains so severe as to lead the patient to commit suicide two years later.

Meynert attributed the abnormal position assumed by the limbs in his patients to disturbance of the muscular sense, a view which is also shared by Nothnagel. Jackson records a case of softening of the right optic thalamus in which the patient could not distinguish between balls of different weight placed in his left hand. As the loss of muscular sense in these cases is always associated with impairment of cutaneous sensibility, the recorded facts do not, in my opinion, justify the conclusion that the optic thalamus stands in any special relation to the so-called muscular sense, whether this be taken to signify the power of appreciating weights or the ability to recognise passive movements communicated to the affected limbs.

Affections of the special senses.—With general hemianæsthesia the special senses are occasionally affected by lesions invading the optic thalamus. This is especially so in tumours. Of the special senses vision is most apt to suffer, and the affection is usually of the homonymous hemiopic character, such as results from lesion of the visual centres or optic radiations. Thus in thirteen cases of tumour of the optic thalamus which I have examined, hemianopsy was present in six, absent in three, and not mentioned in four.

Turner and I found that destruction of the left optic thalamus in a monkey (after division of the corpus callosum and exposure of the ventricle) caused total blindness of the right eye lasting for about a week, followed by right hemianopsy which persisted until the death of the animal some months later. There was no obvious injury to the corpus geniculatum externum, but the optic radiations were degenerated. I cannot find any clinical case in which similar symptoms occurred. But in one recorded by Kirilzew, of glio-sarcoma of the left optic thalamus with hæmorrhagic extravasations in the internal capsule, there was double amblyopia without hemianopsy; and a similar condition was observed by Jakob in a case of extensive destruction of one optic thalamus.

In the majority of stationary lesions of the optic thalamus vision does not seem to have been impaired. And yet the obvious origin of some of the fibres of the optic tract from the pulvinar, and the secondary atrophy which ensues in it when the visual centres are destroyed, furnish grounds for assuming that lesions in this region may cause hemianopsy independently of lesion of the optic radiations or external geniculate body. Several instances of hemianopsy in relation with lesion of the pulvinar have been put on record, namely, by Jackson, Gowers, Edinger, and Henschen.

Against these, however, must be set the cases reported by Zacher and Mills. In Mills' case there was a hæmorrhagic cyst which had destroyed about two-thirds of the substance of the optic thalamus, including the entire external tubercle and a large portion of the pulvinar; and yet there was no hemianopsy. In Zacher's case there was almost complete degeneration of one pulvinar, also without hemianopsy. Henschen is of opinion, from his examination of all the recorded cases of hemianopsy in connection with lesion of the optic thalamus, that no relation can be considered as proved between hemianopsy and lesion of the optic thalamus as such. Nor are the facts sufficient to indicate the precise relations of the pulvinar, external geniculate body, or corpora quadrigemina in the visual scheme.

As regards affection of the other forms of special sense there are comparatively few observations. In a case reported by Hughlings Jackson hearing was equal on both sides, but taste was slightly diminished on the opposite side. The patient in smelling sniffed with one nostril only, but, as he remarks, this may have been due to the general anæsthesia rather than to any affection of smell proper. In a case of tumour of the right optic thalamus under my own care, hearing on the left side was much diminished; smell and taste were, however, normal. In a case reported by Henschen of hæmorrhage in the left pulvinar, hearing in the right ear was defective; and in Zenner's case of tumour of the left optic thalamus hearing in the right ear was not so acute as in the left. In Zenner's case there were also subjective olfactory sensations, and smell in the left nostril was not so acute as in the right. In a case reported by Engel of a large gumma growing from the back of the fornix, and extending over both optic thalami, there was abolition of smell and taste, then of hearing, and lastly of sight. In none of these cases, however, can implication of the internal capsule be excluded.

The relation of lesions of the optic thalamus to **vaso-motor** and **thermal** disturbances is very uncertain, and they have no diagnostic significance. The experiments of Hale White and of Ott on the influence of lesions of the optic thalamus on the temperature of the body are neither harmonious nor conclusive. White, on puncturing the optic thalami in rabbits, at first observed considerable elevation of temperature on both sides of the body; but from later experiments he came to the conclusion that the rise in temperature, which at most was only slight (0·9 F.), was really due to lesion of the corpus striatum. Ott found that puncture of the anterior part of the optic thalamus caused a rise in temperature, but he has since come to the conclusion that the puncture must enter the tuber cinereum, which he regards as the true centre of thermotaxis. Turner and I observed no affection of temperature on destruction of the optic thalamus in a monkey. An examination of the recorded clinical facts does not, in my opinion, show any constant relation between lesions of the optic thalamus and changes in temperature, or disturbance of vaso-motor innervation.

Wharton Sinkler has recorded a case of softening in the posterior part of the right optic thalamus in which death took place from profuse intestinal hæmorrhage without any apparent ulceration of the intestinal mucous membrane. He connects the intestinal hæmorrhage with lesion of the thalamus; and in support of this view he quotes the experiments of Brown-Séquard, Lussana, and others, who found that when lesions were made in the optic thalami ecchymosis occurred in the mucous membrane of the stomach and colon. If, however, there were any real relation between the phenomena we should expect it to occur more frequently, seeing that lesions of the optic thalami are by no means uncommon.

Summary.—(i.) Lesions of the optic thalamus cannot with certainty be distinguished from those involving the sensory division of the internal capsule and optic radiations.

(ii.) Paralysis of the mimetic movement of the face, and the occurrence of post-hemiplegic choreic disorders, are in favour of lesion implicating the opposite optic thalamus.

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LESIONS OF THE CRURA CEREBRI.—The crura cerebri are the connecting link between the cerebral hemispheres and the lower centres, and form the paths by which sensory impressions are conveyed upwards and motor impulses transmitted downwards. Each crus is composed of two parts—a posterior, or dorsal, forming the tegmentum; and an inferior, or ventral, termed the "foot of the crus"; the two being separated by a layer of gray matter termed the "substantia nigra." The exact relations of the various centres and tracts which constitute the tegmentum, substantia nigra, and foot are as yet very imperfectly determined; but there is good reason to believe that the sensory tracts ascend to the cerebral hemisphere in the tegmentum: it is also certain, from embryological investigations as well as by a study of secondary degenerations, that the pyramidal tracts which convey impulses from the motor convolutions are situated in the foot of the crus, and more particularly in the middle third or middle two-fourths of this structure. That portion of the crus which lies external to the pyramidal tracts, which was at one time supposed to be the path of the sensory tracts from below, has been shown by recent investigations to contain fibres which connect the occipital and temporal lobes with the gray matter of the pons, and probably indirectly with the cerebellum. The region internal to the pyramids consists of tracts which descend from the lenticular nucleus and frontal convolutions, and include those which form the anterior division and knee of the internal capsule. In these are contained the tracts from the tongue and facial centres. Internal or mesial to these are fibres which, according to Flechsig, as well as according to Turner and myself, descend from the prefrontal regions; though this is doubted by Zacher, who traces them from the lenticular nucleus. All the tracts, except those which form the pyramids, appear to end in the gray matter of the upper part of the pons.

The crura cerebri are subject to lesions of various kinds. Hæmorrhage limited to the crus is comparatively rare. The same is true of thrombotic softening, dependent on obliteration of some of the branches of the posterior cerebral artery. Abscess is occasionally found invading the crus as well as the optic thalamus and pons. Tumours in the crus, as such, are mostly tuberculous nodules. More frequently the crura are invaded by tumours growing from the base of the skull. Most of the lesions described have been found in the foot or ventral part, and we know more of the symptoms caused by lesions here than in the tegmentum, to which they are very rarely limited.

The complex of symptoms which is characteristic of crus lesion is an alternate hemiplegia involving the limbs, and often also the lower facial region on the opposite side, and the muscles supplied by the third nerve on the same side. When this combination occurs simultaneously the diagnosis of a lesion of the crus is certain. It is less certain if the hemiplegia occur first, and the oculo-motor paralysis at a later date or conversely, though several cases of this kind are on record (Putawski and Goldscheider).

The affection of the third nerve is nearly always complete, involving both external and internal muscles of the eyeball. Thus there is ptosis, which is often the most prominent and earliest ocular symptom, external strabismus with diplopia and giddiness, and mydriasis. Occasionally the oculo-motor paralysis is only partial, and in particular affects the levator palpebræ or the internal rectus. Cases have been observed in which the iris has escaped (Gowers). A superficial lesion may cause oculo-motor palsy without hemiplegia by damaging the root fibres of the third nerve; and partial lesions in the middle line, beneath the corpora quadrigemina, may cause symptoms of acute nuclear ophthalmoplegia.

A tumour growing from one crus may also invade the third nerve on the other side, and thus cause a double oculo-motor palsy. So also the limbs of both sides may be affected by a tumour primarily affecting one crus, and gradually encroaching on the other; and Weissmann has recorded a case in which an intra-peduncular hydatid caused complete paralysis of all four limbs, without paralysis of any of the cranial nerves. In connection with tumours, irritative symptoms may occur in the form of spasms and tremors of the paralysed limbs.

Along with motor hemiplegia there may be impairment or loss of sensation. This would appear to occur more particularly when the lesion extends into the tegmental region of the crus; yet cases have been recorded in which lesions have been found in the tegmentum without causing any noteworthy affection of sensation. Thus Griewe has recorded a case of tumour in the right tegmentum cruris, extending to the optic thalamus in front and the corpus quadrigeminum behind, which, though giving rise to weakness of the left side, caused no oculo-motor palsy or obvious loss of sensation; the patient complained, however, of pins and needles at the tips of the fingers. Krafft-Ebing has related a similar case. Yet this does not negative the view that the tegmentum contains the sensory tracts, as it is well known that tumours may compress the tracts in which they occur without entirely destroying them. In Weber's well-known and much-quoted case of a large focus of hæmorrhage primarily situated in the inner part of the crus, there was considerable loss of sensation—in all probability the indirect effect of pressure on the sensory tracts in the tegmentum. A case of a similar nature has been reported, by Mayor, in which the lesion was also limited to the inner half of the crus. There was motor but no sensory paralysis.

Besides the motor and sensory symptoms above described, there have been cases in which the affection of the opposite limbs was of an ataxic character. Thus Krafft-Ebing has recorded a case of tubercle situated in the tegment of the right crus, which caused left hemiataxy; and he quotes similar cases reported by Buss and Kahler and Pick.

Blocq and Marinesco observed a case of tubercle of the right crus, specially implicating the locus niger, which caused tremors of the left limbs, especially of the arm, similar to those of paralysis agitans, but increased on voluntary movement; and Sachs also has recorded a case of thrombotic softening of the crus, which caused wild ataxic movements of the left arm when voluntary movements were attempted.

In lesions of the crus cerebri the special senses are, as a rule, unaffected, or if so only indirectly; a fact which serves to discriminate such lesions from those due to implication of the internal capsule. The immediate contiguity of the optic tract would lead one to expect occasional hemianopsy, but I have not been able to discover any recent case in which this occurred from crus lesion.

The views of Budge and Afanasieff as to the relation of the crus to the functions of the bladder are not supported by clinical observations; but Nothnagel quotes cases which show that vaso-motor disturbances in the paralysed limbs, in the form of œdema with increased temperature, are not uncommon.

Summary.—Simultaneous paralysis of the third nerve on one side, and of the limbs on the other, is pathognomonic of lesion of the crus cerebri.

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LESIONS OF THE CORPORA QUADRIGEMINA.—The anterior tubercles or nates are connected with the optic tracts by means of the anterior brachia and corpora geniculata externa. The posterior tubercles or testes are connected by the posterior brachia with the corpora geniculata interna, and thus indirectly with the cortex of the temporal lobe. The inferior connections of the anterior tubercles are mainly through the mesial fillet, by means of which each tubercle is connected directly with the nuclei of the posterior columns on the opposite side. Those of the posterior tubercles are chiefly through the lateral fillet. Turner and I (7) have found that section of the lateral fillet, including destruction of its nucleus, is followed by degeneration upwards into the posterior quadrigeminal tubercle on the same side, and downwards to the lateral medullary tract and spinal cord. By means of the lateral fillet also the posterior tubercle is connected with the cochlear root of the auditory nerve, the corpus trapezoides, and superior olivary body; for division of the auditory nerve between the accessory ganglion and the side of the pons causes degeneration in the corpus trapezoides and superior olives on both sides, together with bilateral degeneration of the lateral fillet, more marked on the opposite side.

Diseases affecting the corpora quadrigemina are rarely confined to these structures, and localised hæmorrhage or softening is practically unknown. The most common lesion is tumour, but even tumours occur in this situation more rarely than in other parts of the brain. In all I have been able to discover about thirty cases of tumour of the corpora quadrigemina on record. A unique case of traumatic lesion by a bullet has been recorded by Eisenlohr. In the majority of cases of tumour of the corpora quadrigemina the neighbouring parts are also more or less implicated; especially the optic thalamus, tegmentum pontis, the superior cerebellar peduncles, and often the cerebellum itself. Hence, in the complex of symptoms so caused, it is doubtful how much is due to lesion of the corpora quadrigemina as such, and how much to disturbance of function of the other structures mentioned. But, while this is so, there are certain symptoms, or rather an assemblage of symptoms, which are suggestive of tumour in the corpora quadrigemina or the immediate neighbourhood. These are, as Nothnagel has pointed out, first, an unsteady reeling gait, especially if this appears as the first symptom; secondly, ophthalmoplegia, not quite symmetrical or implicating all the ocular muscles in equal degree.

First, as to gait.—This is usually described as being that characteristic of a drunken person. It resembles that of cerebellar disease rather than ataxy, though in a case reported by myself (5) the opposite leg was lifted high and thrown forwards in an ataxic manner. Nothnagel found this symptom in all cases of tumour implicating the corpora quadrigemina examined by him, except in one recorded by Gowers, in which the lesion was partial and affected only the anterior tubercles (9). It was present in all the cases that have been recorded since 1889, except in one described by Ruel in which the gait was not mentioned; and in another by Guthrie and Turner in which the patient, when he came under observation, was unable to stand; though probably his gait had been of the usual character in the early stages of the disease.

As the disease advances, walking or even standing becomes impossible; but even when this occurs there is no real motor paralysis except when the disease in its growth presses on the motor tracts of the internal capsule or crus cerebri.

The reeling gait and unsteadiness of equilibrium have been attributed by some to affection of the cerebellum itself, or of its superior peduncles, rather than to affection of the ganglia themselves; but the experiments of Rolando, Serres, Cayrade, and Goltz on the lower animals, as well as my own on monkeys, render it probable that lesions of the ganglionic masses of the corpora quadrigemina themselves are sufficient to induce these disorders (6). Nothnagel (11) arrives at essentially the same conclusion from an examination of the clinical cases. The reeling gait is not, however, pathognomonic, as it occurs also in disease of the cerebellum and in hydrocephalus; but if, as Nothnagel remarks, it occurs as the first symptom, and especially if it coexist with oculo-motor disturbances, it points rather to affection of the corpora quadrigemina than of the cerebellum itself.

As a rule the reeling gait occurs before the oculo-motor paralysis; but in some recorded cases the ocular symptoms, especially ptosis, showed themselves first (Taylor, Ransom, and Bruns).

Secondly, as to the oculo-motor symptoms.—Nothnagel has never seen complete ophthalmoplegia in cases of tumour of the corpora quadrigemina. There was, however, complete external third nerve palsy in a case reported by Goldzieher; nearly complete double external ophthalmoplegia in Taylor's case; and in Bruns' case there was double oculo-motor paralysis, external and internal. The most common oculo-motor symptoms are, first, ptosis on one or both sides; secondly, limitation or loss of the upward movements; thirdly, limitation of the lateral movements. In a case reported by myself the upward and downward movements were good, but there was limitation of the lateral movements. In the case of bullet wound reported by Eisenlohr there was divergent strabismus with defective internal movement of the left eye, the wound being on the right side. In Ransom's case there were constant slight clonic movements of the upper eyelids and of the internal recti.

Paralysis or paresis of convergence is not uncommon, as in the cases reported by myself, Bruns, Bristowe, Kolisch, and others. The sixth nerve may entirely escape; but in some cases there is paralysis or paresis of one or both external recti. Thus in Ransom's case the left eye was turned strongly inwards, and the right eye also, but to a lesser extent. Nystagmus does not appear to be common, but it was noted in my own and Taylor's cases. The oculo-motor symptoms are not due to lesion of the ganglionic structure of the corpora quadrigemina, but to implication of the subjacent oculo-motor nuclei. There were no oculo-motor symptoms in a case reported by Nothnagel, nor in another by Weinland.

Irritation of the corpora quadrigemina on one side causes dilatation of both pupils, especially of the opposite one. There appears, however, to be no definite or constant relation between lesions of the corpora quadrigemina and the state of the pupils. The pupils may remain active while there is almost complete external ophthalmoplegia, as in the cases reported by Taylor, Goldzieher, Bristowe, and others. The pupils in some cases react sluggishly. In the case reported by Eisenlohr the reaction to light was lost in both eyes. In that by Guthrie and Turner the light reaction was also probably lost. In Ruel's case the pupil on the side of the tumour was dilated and immobile, and at a later date both pupils became inactive both to light and accommodation. In my own case there was loss of the power of convergence; the pupils were equal and contractile to light, but did not contract on attempts at accommodation. In Weinland's case, in which the tumour was situated on the left side, the pupils were dilated, the right being larger than the left; the reaction to light was retained, but on convergence the reaction was feeble on the right side. The pupils are in may cases dilated or unequal.

Affections of vision.—The obvious connection of the anterior tubercles of the corpora quadrigemina with the optic tracts, and the secondary atrophy which ensues when the corresponding optic tracts are divided, or the cortical centres of vision destroyed, show these bodies to be important parts of the visual apparatus; but their exact relations to the sense of vision are not clear. Many experimenters have found that destruction of the optic lobes, or anterior corpora quadrigemina, causes total blindness; but these results receive little support from the facts of clinical observation. Dr. Bastian mentions a case of total blindness apparently due to a patch of softening almost limited to the anterior quadrigeminal bodies. Nothnagel, however, as the result of his examination of reported clinical cases, makes the statement that vision or visual acuity may be unimpaired although the corpora quadrigemina be entirely destroyed. In Eisenlohr's case of bullet wound of the right corpora quadrigemina, there was no affection of vision immediately after receipt of the injury; but three months later vision in the right eye was diminished, and at a still later date visual acuity was diminished in both eyes. The probability is, therefore, that the defective vision was due to secondary changes in the optic nerves, as otherwise the affection of vision should have occurred on receipt of the lesion.

In almost all the cases of disease of the corpora quadrigemina, in which vision has been defective, the lesion has been of the nature of tumour, with coincident optic neuritis or internal hydrocephalus. In Taylor's case, however, there was no optic neuritis, and yet the child was blind; and Dr. Ransom is of opinion that in his case the defective vision was greater than could be accounted for by the neuritis. It should also be remembered in this connection that there may be perfect vision, notwithstanding the existence of considerable neuritis; as was pointed out long ago by Hughlings Jackson. Defective vision from optic neuritis usually sets in only when the inflammation gives place to secondary atrophy. In most cases, however, of unilateral lesion of the corpora quadrigemina, with defective vision, there is little or no difference in the visual acuity of the two eyes; and this increases the probability that the true cause is either optic neuritis or internal hydrocephalus.

It is obvious from these facts, therefore, that a direct relationship between the anterior quadrigeminal tubercles and the sense of vision is far from being established by clinical observation.

Affections of hearing.—Experimental observations, as well as a study of the course of degeneration in lesions of the temporal lobe (p. 319), render it probable that the central tracts of the cochlear nerve ascend through the opposite lateral fillet, posterior tubercle, brachium and internal geniculate body, and thence to the internal capsule. It is important, therefore, to examine whether there are any evidences of affection of hearing in connection with diseases of the corpora quadrigemina. Weinland has analysed the clinical records in this relation, and finds that in nineteen cases of tumour of the corpora quadrigemina hearing was affected in nine; and of these, five had impairment of hearing on both sides. In three cases (reported by myself, Weinland, Ruel) hearing was impaired on the side opposite the tubercles affected; and Weinland concludes, therefore, that disease of the posterior corpus quadrigeminum causes affection of hearing on the opposite side. There are, however, many difficulties in the way of arriving at a correct conclusion on this point; chiefly dependent on the mental condition of the patients, who usually present a considerable degree of mental dulness, somnolence, and apathy. Hence impairment of hearing, even if observed, may be attributed to this cause; or, unless very pronounced, it may altogether escape observation.

Affections of motion and sensation.—As a rule, in lesions of the corpora quadrigemina there is no motor paralysis or loss of sensation; and where such exists it may be referred to direct or indirect implication of the motor or sensory tracts of the cerebral peduncle or internal capsule.

Tremors, however, are not uncommon, chiefly of the arms; and especially on volitional movement, like those of disseminated sclerosis. Tremors of this character were observed in ten out of seventeen cases I have analysed (*vide* cases by Ferrier, Weinland, Bruns, Ilberg, Bristowe, Kolisch, Taylor, Eisenlohr, Pilz, Guthrie and Turner). In five of these there was also some degree of motor weakness; in the others this was not observed. The tremors were either bilateral, or, if unilateral, on the side opposite the lesion, except in Weinland's case. In those cases in which the tremor was bilateral the lesion was not strictly confined to one side. Oscillation of the head and neck, as well as of the limbs, was noted in the cases reported by Bristowe and by Taylor. In Ilberg's, and also in Eisenlohr's case, the tremors were described as being similar to those of paralysis agitans.

An examination of the cases, in which an exact necropsy was made, shows that in all in which tremors were observed, the lesion was not confined to the corpora quadrigemina, but implicated also the optic thalamus, tegmentum cruris or pontis, or superior cerebellar peduncles; and it is a question, therefore, how far the tremors depend on the corpora quadrigemina or on the coincident lesions. In this connection it is worthy of note that Turner and I have described similar tremors as resulting from section of the superior cerebellar peduncles in monkeys (7). In our experiments, in which the peduncle was divided between the cerebellum and its decussation in the tegmentum, the tremors were confined to the side of lesion. If the tremors observed in clinical cases are in reality due to lesion of the superior peduncles, one might explain the crossed tremor in unilateral lesion by affection of the cerebellar peduncle above its decussation.

Experimental irritation of the corpora quadrigemina gives rise to tonic or tetanic spasm, passing into complete opisthotonos if the irritation be long continued. A condition similar to this experimental result has been described by Dr. Hughlings Jackson in connection with tumours of the middle lobe of the cerebellum. I am inclined to attribute the opisthotonic spasm to irritation of the corpora quadrigemina, rather than to the cerebellum itself. This is supported by Goldzieher's case of tumour of the corpora quadrigemina of the size of a hazel nut, in which there were convulsions and opisthotonic spasms of the limbs.

The knee-jerks are either normal or exaggerated. They may, however, be abolished even at an early date, just as in cerebellar disease. (Guthrie and Turner, Ransom). They were exaggerated in the cases reported by Kolisch, Bristowe, and myself.

In connection with lesions of the corpora quadrigemina it is only necessary to allude in this place to paralysis of the ocular muscles, internal and external, which occurs from lesions limited to the oculo-motor nuclei, as the subject is discussed elsewhere at length (vol. vi. p. 779). These are, with the exception of the nucleus of the sixth, situated in the central gray substance subjacent to the aqueduct of Sylvius, and consist of different cell groups which have probably special relations to the various internal and external ocular muscles, and are connected with the sixth nuclei by the posterior longitudinal bundles. Lesions of the oculo-motor nuclei may be acute or chronic. The most common lesion is a chronic degeneration of the nerve cells, similar to that which occurs in bulbar palsy and progressive muscular atrophy, and is mostly associated with or followed by symptoms of tabes. The oculo-motor palsy may be limited to the internal muscles, or affect some or all of the external muscles, so that the condition is one of partial or complete ophthalmoplegia interna as well as externa. An acute form is more rare, and in its onset and pathology resembles acute anterior poliomyelitis. From its analogy with this affection Wernicke has named it acute polio-encephalitis superior—the term superior being used to distinguish it from a similar affection of the nuclei of the medulla oblongata, which is named acute polio-encephalitis inferior. In these cases ophthalmoplegia occurs suddenly with ptosis and paralysis of the conjugate movements of the eyes, together with loss of convergence and the power of accommodation. Such were the symptoms in a case recently under my care which ended in recovery in the course of a few weeks, with the exception of the powers of convergence and accommodation. The reaction of the pupils to light was sluggish, but not abolished; several similar instances have been recorded. A third form of lesion, termed "sudden nuclear palsy," has been attributed to obstruction of the branches of the basilar artery which supply the oculo-motor nuclei. The oculo-motor paralysis in this case is usually irregular, and associated with some degree of hemiplegia, especially on the side opposite the greater lesion.

Summary.—A regional diagnosis of lesion of the corpora quadrigemina is probable when there is a combination of ophthalmoplegia with an uncertain or reeling gait. The probability is increased if there is also some affection of hearing in one or both ears.

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Lesions of the pons Varolii.—Lesions of the pons Varolii are especially diverse in their symptomatology owing to the complex relations of the pons itself. Not only is it an independent centre, but it contains the sensory and motor paths to and from the cerebrum; it is intimately linked with the cerebellum by means of the middle peduncles, and through it pass many of the cranial nerves in their courses to or from their primary nuclei. The symptoms are therefore likely to vary greatly according to the position and extent of the lesion. To give an explanation of these in all their relations necessitates a knowledge of the minute anatomy of the pons, which it is necessary for the purposes of this article to assume on the part of the reader.

We may, however, somewhat simplify the symptomatology of lesions of the pons by classifying them with Markowski according as they affect the *tegmental* or *crustal* region. Lesions of the tegmental region are specially apt to affect the fifth, sixth, seventh, and eighth nerves, as well as to cause disturbances of sensation and co-ordination; lesions of the crustal region affect principally the movements of the limbs, as well as those supplied by the seventh, tenth, eleventh, and twelfth cranial nerves.

The pons is liable to various morbid processes, originating in it primarily or affecting it secondarily. Of the latter character may be specially mentioned aneurysm of the basilar artery, and tumours of the cerebellum or base of the skull.

Hæmorrhage is by no means uncommon, though it occurs much less frequently here than in the central ganglia. The hæmorrhagic extravasation may vary in size from a hemp-seed to one which causes complete disorganisation of the whole structure. Occasionally there may be several small hæmorrhages quite distinct from each other (Russell and Taylor). Large hæmorrhages not infrequently burst into the fourth ventricle, or even make their way through the iter into the lateral ventricles. Small hæmorrhages into the pons are not necessarily accompanied by a loss of consciousness; but if the hæmorrhage be large, profound coma occurs with great suddenness, and leads to death much more certainly and rapidly than similar effusions in other parts of the cerebrum. Death takes place in a few minutes (7 minutes, Mickle); or in a few hours (2 hours, Rorie; 4½ hours, Barr). Bode finds that of 78 cases 46 died within twenty-four hours.

In pontine hæmorrhage, along with profound coma, there is often complete relaxation of the muscular system and contraction of the pupils, so that the condition is not unlike that found in opium poisoning. Tonic or clonic convulsions sometimes occur, rarely unilateral, and often irregular in type and distribution. The occurrence of paralytic or convulsive symptoms in the limbs of one side and the face on the opposite is pathognomonic of hæmorrhage in the pons. When the limbs are paralysed on one side there is not infrequently conjugate deviation of the head and eyes from the side of the lesion; but if the symptoms are convulsive the deviation of the head and eyes may be towards the side of the lesion. Deglutition is difficult or impossible, and death occurs from cardiac or respiratory paralysis; irregularity of rhythm generally preceding the fatal issue. Hyperpyrexia is frequently observed, and the temperature may rise as high as 109° F. Hæmorrhage may occur in the pons not only from diseased arteries but, as has been shown experimentally by Duret, from cranial injuries also, more particularly in the frontal region; and I have seen several cases of cranial injuries in man in which the symptoms were those of lesions of the pons, in all probability of a similar nature.

Necrotic softening.—The most common cause of necrotic softening of the pons is thrombosis, due to atheromatous or syphilitic degeneration of the basilar artery or its branches. Embolic softening is exceedingly rare, though a case of this kind has been reported by Gowers. If, however, as Nothnagel points out, an embolus sticks in the vertebral, the basilar artery may become gradually thrombosed and blocked.

Tumours occasionally grow in the substance of the pons, but more frequently the pons is pressed on by tumours originating at the base, or situated primarily in the cerebellum. Sometimes the whole pons may be enlarged by gliomatous infiltration, constituting so-called hypertrophy of the pons.

Abscess is rare, but the pons is a favourite seat of sclerotic foci, as in multiple sclerosis; though in this latter case the pons is only affected along with other encephalic centres.

Affections of motion.—As a rule in lesions of the pons affections of motion predominate over affections of sensation. They may be variously grouped, and at the outset it may be mentioned that cases of lesions of the pons have been recorded in which no symptoms were noted either in the domain of motion or sensation. A remarkable instance of this kind has been recorded by Ladame, in which the whole pons appears to have been transformed into a new formation containing no nerve elements, and covered over only by a layer of medullated fibres a line thick.

Type 1.—The limbs may be paralysed, but the cranial nerves and nuclei escape. In this case the lesion is generally in the upper part of the pons, and the resulting hemiplegia is indistinguishable from that due to lesion of the internal capsule.

Type 2.—The cranial nerves or nuclei may be affected alone without paralysis of the limbs. In Bennett and Savill's case of softening in the region of the sixth nucleus there was isolated conjugate deviation of the head and eyes. In Mierzejewski and Rosenbach's case of glioma there was right facial palsy and conjugate deviation to the left. In Moeli and Marinesco's case there was paresis of the lower half of the right side of the face, the right sixth, and right portio minor of the fifth, together with affection of sensation on the left side of the body. In Elzholz's case of hæmorrhage there was complete paralysis of the right sixth, seventh, and of the twelfth, without palsy of the limbs, a rare condition in hæmorrhage.

Type 3.—There may be paralysis of the face and limbs on the side opposite the lesion. In this case the lesion must be above the decussation of the central facial fibres, which is probably about the middle of the pons (see cases by Diller, Mills and Zinner, Markowski). The resulting hemiplegia may be indistinguishable from that due to lesion of the internal capsule, unless some localising symptom be present, such as implication of the fifth on the side of lesion, or of the sixth, as in Mills' and Zinner's case.

In ten cases which have come under my observation five showed this form of paralysis.

Type 4.—The most characteristic form of paralysis due to lesion of the pons is paralysis of one or more of the cranial nerves, especially the fifth, sixth, and seventh on the side of lesion, and of the limbs and tongue on the other side. This is the type of the so-called alternate hemiplegia (Grubler).

The most common variety of this alternate hemiplegia is paralysis of the face on the side of lesion, and of the limbs on the other. The facial paralysis is of the peripheral type, and implicates both the upper and lower facial regions; the paralysis of the limbs is of motion alone, or of this combined with anæsthesia. In order to cause this group of symptoms the lesion must be below the decussation of the facial fibres; that is, below the middle of the pons. This grouping of symptoms is practically pathognomonic of pons lesion, especially if it occur suddenly. It may, however, be simulated by a basal tumour, or chronic meningitis, compressing the pons and facial nerve; but in this case the onset is usually slow. Joffroy has seen similar symptoms produced by a double cerebral lesion.

(*a*) The seventh may be the only cranial nerve affected on the side of lesion, but more often other nerves suffer, especially the sixth and fifth. Occasionally, but more rarely, the eighth is also implicated. If the lesion extend into the medulla oblongata, the eleventh and twelfth cranial nerves may likewise become affected, as shown by unilateral paralysis of the palate, vocal cord, and tongue; and in some cases the ninth and tenth have been involved. Thus we find the seventh and sixth (Porter, Williams); the seventh, sixth, fifth (motor and sensory), and twelfth (Jolly); the seventh, sixth, fifth (motor and sensory), and palate (Bristowe); the seventh, fifth, and eighth (Miles); the seventh, fifth, and eleventh (vocal cord and palate) (de Havilland Hall); and among my own cases I have seen the seventh, sixth, fifth, eleventh (vocal cord and palate), twelfth, and eighth; and also a case of seventh, sixth, fifth, and partially also the opposite sixth. Oppenheim records a case in which tubercle of the left side of the pons caused paralysis of the left side of the face, left sixth and fifth, right internal rectus, right side of the face in the lower part, and the right limbs. In cases recorded by Martin Brasch and Mills and Zinner the upper part of the face only was affected, on the side of lesion. This is rare. Though in both cases the focus seems to have affected the issuing seventh, as there was also conjugate paralysis the lesion may have implicated the posterior longitudinal bundle, in which it is probable that the upper facial fibres from the third nucleus descend to the genu of the facial nerve. One side of the face may be paralysed and the other in a state of spasm.

(*b*) Next in frequency to affections of the seventh cranial nerve and limbs is paralysis of the sixth. It is often, as we have seen, affected along with the seventh; but the seventh may escape, as for example in the case of Blocq and Guinon. Usually the hemiplegia, associated with affection of the sixth nerve, does not involve the face; but Raymond records a case of supposed pons lesion in which there was affection of the sixth on one side, and the face and limbs on the other; and a similar case has been reported by Mills and Zinner. This is a rare form of alternate hemiplegia. There are also cases of paralysis of the face, sixth nerve, and limbs on the side of lesion. When the root of the sixth nerve is implicated there is paralysis of the abducens on that side, with internal strabismus; but if the nucleus of the sixth is affected, there is loss of conjugate movements of the eyes towards the side of lesion, if it is destructive; and conjugate spasm to the side of lesion if it is of an irritative character. In all the cases that have been reported of this affection, the lesion has been in the sixth nucleus, or immediately above it, except in one case reported by Senator, in which the lesion was just below the lower end of the sixth nucleus. The most probable explanation of this conjugate deviation from unilateral lesion of the sixth nucleus is that the cortical fibres for the opposite external rectus, and for the internal rectus of the same side, run down together into the region of the sixth nucleus of the opposite side, having decussated higher up, perhaps in the region of the corpora quadrigemina. Of these fibres some enter the sixth nucleus, and others ascend by the posterior longitudinal bundle into the third nucleus of the same or the opposite side. It is usually assumed that the fibres which ascend in the posterior longitudinal bundle cross over to the opposite third nucleus; but it is unnecessary to assume this decussation if, as Kölliker and Perlia hold, there is a partial decussation of the fibres of origin of the third nerve in the middle line. That the posterior longitudinal bundle contains fibres, interruption of which causes paralysis of the opposite internal rectus, may be regarded as well established, both by the results of experiment and also from clinical cases. Thus Grubler records a case of tubercle on the left side, immediately under the anterior segment of the floor of the fourth ventricle, which caused paralysis of the right internal rectus without implication of the left abducens. As a rule, in these cases of conjugate deviation of the eyes from lesion of the sixth nucleus, the power of convergence is retained.

(*c*) Affections of the fifth nerve are, as we have seen, very common on the side of lesion. The sensory division seems to suffer more than the motor, but not infrequently the two are affected together. The sensory and motor divisions were both affected in the cases reported by Jolly, Bristowe, de Havilland Hall, and in a case of my own; the sensory alone in cases reported by Kolisch, Brasch, Starr, Holmberg, Miles; and the motor alone in the case reported by Moeli and Marinesco, and in another case under my own care. The region of the skin supplied by the fifth usually escapes on the side of the hemiplegia, but it was affected in Bristowe's case, in which there was complete hemianæsthesia, including the face.

Tactile sensibility may be impaired in the region of the trigeminus on the side of lesion, and on the limbs of the opposite side; and thus we get crossed hemianæsthesia, as in the cases reported by Jolly and Miles. The trigeminal anæsthesia affects either the whole of the region innervated by the fifth, or only that supplied by special divisions. Thus in the cases reported by Bristowe, Holmberg, and Brasch, the affection of the fifth was limited to the upper area of distribution. Along with the face, the conjunctiva, nostril, and tongue are often anæsthetic. Cases have been reported of pons lesion in which tactile sensibility in the region of the fifth was normal, but in which there have been complete analgesia and thermanæsthesia (Starr, Holmberg).

In some cases not only is the eye on the side of lesion anæsthetic, but it may become the seat of neuro-paralytic ophthalmia, as in the cases reported by Bristowe, de Havilland Hall, Miles, and Starr. It is, however, a question whether the trunk of the fifth was not affected in these cases, as the neuro-paralytic ophthalmia is more common in peripheral nerve lesions. When it does occur we may, according to my own and Turner's experiments (13), assume the existence of an irritative lesion, and not the cessation of a trophic influence.

Taste may be impaired on the side of the trigeminal anæsthesia, as in the cases reported by Kolisch and Bristowe. But in de Havilland Hall's case, though there was complete paralysis of the fifth, both motor and sensory, taste was normal. Instead of anæsthesia of the fifth there may be hyperæsthesia or neuralgic pains. Thus in de Havilland Hall's case the earliest symptom was pain referred to the malar branch of the fifth, and the eye became so painful and inflamed as to require enucleation.

Affection of the motor division of the fifth is shown by paralysis and wasting of the muscles of mastication. In some cases, however, there may be irritative symptoms in the form of trismus.

When the tongue is affected it is usually on the side of hemiplegia, though it frequently escapes. If the lesion extend down to the medulla, so as to implicate the roots or nucleus of the twelfth, the tongue is affected on the side of lesion. Madame Goukovski records a case of paralysis of the limbs and tongue on the opposite sides, due to softening in the left olive and pyramid, which also affected the roots of the twelfth nerve; a condition of things readily understood from the anatomical relations of these structures.

Bilateral symptoms occur frequently in lesions of the pons, either acute or chronic. Bilateral paralysis is especially common in hæmorrhage, and it may affect either the arms or legs or both; though bilateral paralysis of the legs is very rare. Bilateral paralysis is also common in cases of thrombotic occlusion of the basilar artery. In a case reported by Hoppe there was, at first, left hemiplegia, with double paralysis of the sixth and seventh cranial nerves, and twenty-four hours later also right hemiplegia. When the occlusion is confined to the branches of the basilar artery we may, as Gowers has pointed out, get successive and irregular paralysis of the limbs and cranial nerves in almost every variety of distribution. In cases of tumour growing to any size in the middle of the pons we may have paralysis of all four limbs and of the tongue, with difficulty of articulation and deglutition, as well as double paralysis of the fifth, sixth, and seventh cranial nerves. At other times the symptoms are quite unsymmetrical. Thus the face may be paralysed on one side, together with all four limbs, the affection being greater on one side than the other; or the limbs may be affected on one side and the face on both. In the case of a tumour originating on the one side the symptoms may at first be unilateral, but ultimately become bilateral with the extension of the disease to the other side. Thus in the case reported by Blocq and Guinon there was at first conjugate paralysis to the right, followed at a later date by bilateral conjugate paralysis. The tumour involved the right sixth nucleus and pressed on the left, and other similar cases have been reported.

Affections of hearing.—Affections of hearing are not regarded as common in cases of lesions of the pons. Nothnagel refers to cases reported by Homberg, Rosenthal, and Huguenin, in all of which hearing was affected on the side of lesion. Of twelve cases of tumour and five of softening of the pons, together with ten others which have come under my own observation, I find that hearing was affected on one or both sides in five. In Kolisch's case (tumour of the right tegmentum) hearing was affected chiefly on the side of lesion. In Bristowe's case (tuberculous mass chiefly in the left side of pons) hearing was affected especially in the right ear. In this case there was left hemiplegia. In Diller's case, in which there was left hemiparesis, hearing was defective on the left side. In Miles' case (tumour in the left half of the pons) hearing was defective on the side of lesion; and in a case under my own care, in which there was hemiplegia and paralysis of several of the cranial nerves on the right side, hearing was defective in both ears, beginning on the right side; that is, three times exclusively, or mostly, on the side of lesion, and twice on the opposite side.

In Jolly's case (tumour on the left side of the pons, just underneath the floor of the fourth ventricle) degeneration was found in the tracts now usually regarded as the central paths of the cochlear nerve; namely, the corpus trapezoides, striæ acusticæ, and crossed lateral fillet. This, according to theory, ought to have been associated with bilateral affection of hearing, which, however, does not appear to have been the case,—a fact difficult to explain, unless we assume that the tumour did not actually destroy the continuity of these tracts.

Besides the actual impairment of hearing, such as occurred in the above-mentioned cases, subjective sensations of sound have also been observed in several instances.

Affections of cutaneous sensation.—Affections of sensation are of frequent occurrence in connection with lesions of the pons. Ladame found hemianæsthesia along with hemiplegia in about one-third of the cases. The hemianæsthesia may include the face and limbs on the same side, which is rare (Kolisch); or the face may be affected on the side of lesion, and the limbs on the other side, which is the common form, according as the lesion is above or below the point of junction of the ascending and descending roots of the fifth nerve (Starr, 44). Or we may get the fifth affected on both sides, and the limbs only on one side, as in Bristowe's case, in which there was anæsthesia of the face on the side of lesion, and hemianæsthesia of the opposite side, including the face. The degree of disturbance of sensation varies from slight diminution up to complete abolition, and all forms of sensibility appear to be about equally affected.

The position of the sensory tracts in the pons is a subject which cannot be said as yet to have been definitely determined. So few cases of limited lesions, accurately investigated, have been reported, that it is difficult to speak with any certainty on the point.

The lesions producing anæsthesia are, according to Nothnagel, usually situated laterally and near the floor of the fourth ventricle. In a later analysis of the recorded cases, Starr (44) by a process of exclusion arrived at the opinion that the sensory tracts in the pons must lie between the deep transverse fibres and the gray matter of the fourth ventricle; that is, in the lemniscus or formatio reticularis.

Moeli and Marinesco, in an exhaustive paper (32), in which they describe one new case and examine all those previously recorded, conclude that in all cases in which the fillet and the ventral part of the formatio reticularis were affected there were disturbances of cutaneous sensibility; and that when these parts were not affected cutaneous sensibility was intact. Whence they argue: "We can localise the paths of cutaneous sensibility either in the mesial fillet alone, or in it together with the neighbouring part of the formatio reticularis." Buss, however, records a case of softening of the formatio reticularis reaching quite up to the fillet, in which tactile sensibility was unimpaired. This case would seem to exclude the formatio reticularis from any share in the conveyance of sensory impressions. And Blocq and Guinon record a case of tumour implicating the fillet in which there was no affection of sensation. Turner and I (13) have found that after destruction of the clavate and cuneate nuclei in monkeys, complete degeneration takes place in the mesial fillet without any affection of cutaneous sensibility, and, indeed, without any notable symptoms except a transient unsteadiness of gait. It is obvious, therefore, from facts like these, that there are serious objections to the view that the mesial fillet is the path of tactile, or, indeed, of any of the forms of conscious sensibility. Our experiments point to the short fibre-systems of the tegmentum, or the formatio reticularis, as being the path of transmission of cutaneous sensation proper. Starr and others have attempted to trace the differentiation of the paths of the different forms of sensation, but for the present we cannot regard this as having been at all satisfactorily made out.

Ataxic disorders.—Ataxic disorders have been frequently observed in connection with lesions of the pons, and Nothnagel is of opinion that these may be attributed to pontine lesions as such, apart from implication of the cerebellum or its peduncles.

Moeli and Marinesco state that in all cases in which ataxy has been a noteworthy symptom, the middle and ventral parts of the tegmentum have been affected. In almost all these cases there were disturbances of cutaneous sensibility; but Buss and Leyden have each recorded a case in which ataxy was well marked, but tactile sensibility apparently unimpaired. Goldscheider insists on the relation of ataxy to disturbance of the muscular sense, and records a case of ataxy with loss of so-called muscular sensibility due to lesion of the interolivary tracts between the roots of the twelfth nerve and the raphé. Reinhold has also recorded a case of focal lesion in the right side of the medulla associated with ataxy of the right arm, which he attributes to implication of the fibres of the fillet arising from the right post-columnar nuclei. Jolly, however, has recorded a case of lesion of these very parts in which there was no ataxy.

It is obvious from the above facts that the pathology of the ataxic disorders occasionally met with in connection with lesions of the pons is in need of further investigation.

The gait has been frequently described as of an uncertain or reeling character, similar to that which is seen in connection with cerebellar disease. Many cases of this kind might be referred to. In Penzoldt's case there was, besides the reeling gait, a tendency to retropulsion; and in Ewald's case there were also pendulum-like oscillations of the limbs. In a case reported by Williams (gliomatous affection of the whole pons) the patient would tumble about, fall forwards, or spin round like a top. These disturbances of station and locomotion may be partly due to vertigo, with which they are often associated; or both symptoms may be due to the same cause, namely, implication of the cerebellum or its peduncles. As nearly all the well-marked cases of this affection have been of the nature of tumour, an indirect influence on the cerebellum must always be assumed as probable. Choreic movements, or jerky tremors similar to those seen in disseminated sclerosis, have also been noted in connection with lesions of the pons, and are probably also indirect effects, not characteristic of pontine lesions as such.

Affections of articulation and deglutition.—Affections of articulation and deglutition, particularly the former, are common in connection with lesions of the pons; they are usually very pronounced in occlusion of the whole basilar artery, as in a case reported by Hoppe.

Impairment of articulation in pons lesion is, without doubt, due to affection of the central fibres of the hypoglossal; but dysphagia, when it occurs, is probably due to indirect implication of the medulla oblongata; or is a secondary result of the impairment of the movements of articulation which form the first part of the act of deglutition.

Anarthria occurs more particularly when the pontine lesion is bilateral. Unilateral lesions of the pons may exist without any impairment of articulation. From an analysis of 27 recorded cases Markowski finds that of 18 of unilateral lesion there were 10 in which there was no dysarthria; of these, 7 were lesion of the right half of the pons, and 3 of the left. In the remaining 8—or, on more critical analysis, 6—impairment of articulation was observed. In 9 cases of bilateral lesion there was only 1 in which anarthria did not occur. The conclusion, therefore, from these facts is that bilateral lesion of the pons almost always, and unilateral in less than one-half of the cases, cause impairment of articulation. The results are the same whether the lesion is in the right or in the left side; so that the view of Wernicke, that the articulatory tracts run specially in the left half of the pons, is not substantiated. The paths of articulation appear, from Markowski's observations, to run specially in the medio-dorsal portion of the pyramid. In his own case, in which there was well-marked dysarthria and dysphagia, there was total destruction of the left pyramid, and only a small focus in the medio-dorsal part of the right pyramid. He quotes a similar case, reported by Raymond, in which there were two small foci in the middle line of the pons which could only have injured the medio-dorsal part of the pyramidal tracts.

Hyperpyrexia.—Many other symptoms occur in connection with pontine lesions, but are not peculiar to them. There is one, however, which deserves special mention, namely, hyperpyrexia. This occurs more particularly in connection with sudden lesions, such as hæmorrhage. The temperature not infrequently rises above the normal within an hour of the onset of hæmorrhage, and speedily rises to 104° or 106°, or even higher, before death. In a case reported by Barr the temperature rose to 109°. A similar rise may occur in sudden obstruction of the basilar artery, preceded by an initial fall, such as not infrequently occurs at the onset of cerebral hæmorrhage. Bastian reports a case of this kind in which there was an initial fall to 95° F. in the rectum. In chronic lesions of the pons, such as tumours, softening, etc., there may be no material alteration of the temperature. In some cases it is actually subnormal.

The pupils.—The state of the pupils has been already specially referred to in connection with the symptoms of hæmorrhage into the pons. In pontine hæmorrhage the pupils are usually minutely contracted, as in opium poisoning. This, however, is not universal, and the state may vary according to the period at which they are observed. Thus in a case reported by Edridge Green the pupils were at first minutely contracted, but they became dilated before death; and they were normal in a case recorded by Rorie. Glycosuria and albuminuria have sometimes been observed in connection with lesions of the pons, more frequently, perhaps, in cases of hæmorrhage. It is doubtful whether any causal relationship has been satisfactorily established between these symptoms and lesions of the pons as such. Lesions of the pons frequently occur in connection with chronic renal disease and arterial sclerosis; so that the albuminuria, in some instances at least, has been pre-existent; and glycosuria may sometimes be found in other lesions of the nerve centres.

Summary.—Lesions of the pons Varolii are very varied in their symptomatology. The most characteristic indications are a combination of paralysis of some of the cranial nerves, especially the fifth, sixth, and seventh, on the side of lesion, and of the limbs on the other.

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Lesions of the Cerebellum.—The cerebellum is liable to the same diseases as affect the other encephalic centres.

Hæmorrhage.—Hæmorrhage occurs much less often in the cerebellum than in the cerebrum, a fact which is in accordance with the relatively greater frequency of miliary aneurysms in the cerebrum. In 100 cases of cerebellar disease quoted by Krauss there was only one of hæmorrhage. In many cases the extravasation is large and occupies the greater portion of one hemisphere, as well as part of the middle lobe.

According to Duret, the great effusions come from the chief stem of the branch of the superior cerebellar artery to the dentate nucleus. Small circumscribed foci of hæmorrhage are comparatively rare. They are for the most part central, and usually implicate the nucleus dentatus. Hæmorrhages restricted to the vermis appear to be exceptional (Nothnagel). Hæmorrhage into the inner and hinder portion of the hemisphere may occur from rupture of a branch of the posterior cerebral artery (Gowers). Hæmorrhagic extravasation may burst into the fourth ventricle and may fill all the ventricles, as in a case reported by Barling. The superior and inferior cerebellar peduncles are rarely the seat of hæmorrhage; but extravasation may occur in the middle peduncle, generally from a hæmorrhage primarily originating in the side of the pons; and conversely a hæmorrhage into the cerebellum may extend into the side of the pons. In 10 cases of cerebellar hæmorrhage, to which I have been able to refer, 5 were in one hemisphere, 3 were in the middle lobe, and also in one or both hemispheres. In Mills' case the cerebellum, pons, and crura were quite ploughed up; and in Campbell's (23) case 3 there was perivascular hæmorrhage in both corpora dentata and in the adjoining white matter. In three cases extravasation had occurred into the fourth ventricle.

Symptoms of cerebellar hæmorrhage.—There may be premonitory symptoms, such as headache and giddiness, for two days before the onset, as in Mills' case; and Ratcliffe has noted, as of probably some significance, an involuntary movement of the head to one side. When the hæmorrhage occurs there is usually coma, as in intracranial effusions generally. Often it is sudden; this is so in half the reported cases, but the coma may be delayed, or, according to the observations of Ratcliffe, may cease and reappear several times. Pain in the head, especially occipital, is not infrequently a marked and early symptom. Vomiting is frequent and often very persistent; the symptom is so marked, that, according to Nothnagel, cerebellar hæmorrhage may be expected if at the beginning there is repeated vomiting without any obvious motor paralysis. But hæmorrhages of considerable extent have taken place without any symptoms during life (Oliver). Hemiplegia may or may not occur. Its absence is generally regarded as more typical than its presence. In the ten cases to which I have referred there was no paralysis in three; crossed hemiplegia in one (Farquharson); direct hemiplegia in Thacker's case; weakness of the arm on the side of lesion, and paresis of the face on the opposite in Friedeberg's case; and left hemiplegia in Mills' case, in which the cerebellum, pons, and crura were all torn up and disorganised. The state of the pupils is somewhat variable. Generally they are contracted and fixed, and in Barling's case they were contracted at first, but became dilated before death. Not infrequently, in connection with hæmorrhage into the middle lobe, priapism has been observed, due without doubt, as Segalas has pointed out, to irritation of the subjacent pons medulla, and not to any direct connection between the middle lobe and the generative organs, as supposed by Serres.

Hæmorrhage into the cerebellum is often very rapidly fatal, with severe respiratory symptoms; especially if effusion take place into the fourth ventricle. If recovery ensues the pressure symptoms pass away, and there remain only, with greater or less persistence, the symptoms specially characteristic of cerebellar lesion.

Softening.—Necrotic softening of the cerebellum from vascular occlusion, embolic or thrombotic, is rare. Even if a cerebellar vessel does become occluded the softening is comparatively limited, owing to the rich anastomosis between the cerebellar vessels. Softening is most common in the regions supplied by the posterior cerebellar artery, often secondary to occlusion of the vertebral, but sometimes originating in the posterior cerebellar, and extending downwards into the vertebral artery, as in a case reported by Menzies.

Abscess.—Abscess frequently occurs in the cerebellum, and, for the most part, in connection with disease of the middle ear. Winter and Deanesly collected 23 cases of cerebellar abscess, and found purulent otitis media in all. In 19, which I have myself analysed, there was middle ear disease in 16. In all these the abscess was situated in the hemispheres. In Winter and Deanesly's collection the abscess was situated in the anterior part of the lateral lobe in 21; in the inferior surface of the middle lobe in 1; and in the flocculus in 1. In a case reported by Friedeberg there was an abscess in each hemisphere.

The time that elapses between the onset of the otitis media and the development of cerebellar abscess is very variable—between three weeks and several years. Cerebellar abscess may also result from traumatic lesion and septic infection, as in a case reported by Krauss.

Tumours.—Tumour occurs very frequently in the cerebellum. Ladame in 1865 had collected 77 cases; Bernhardt between 1865 and 1881 had collected 90 cases; and Wetzel between 1881 and 1889 collected 59 cases. I have collected, mostly since 1890, records of 53 cases, of which 8 are original and unpublished. Besides the 53 cases of tumour proper, 11 cases of cyst might be included. Simple cysts are not common. Most of the so-called cerebellar cysts are merely cystic degeneration of tumours; and Williamson rightly points out that a cerebellar cyst can only be classed as simple or serous when minute examination shows no trace of hydatid disease, cysticercus, hæmorrhage, or tumour. He has himself recorded two cases of apparently simple serous cyst; but histological examination showed gliomatous or glio-sarcomatous tissue in the walls of both, so that they were merely tumours, the proper tissue of which had almost completely disappeared.

Atrophy.—Atrophy of the cerebellum is not uncommon, and varies greatly in degree. The whole organ may be entirely atrophied, as in the much-quoted case of Alexandrine Labrosse, and a similar case, under the care of Dr. Shuttleworth, described by myself (36); or only one, or both hemispheres may be affected, the middle lobe being normal or relatively so. Atrophy of one hemisphere is usually consecutive to long-standing disease of the opposite cerebral hemisphere. Atrophy of the cerebellum may be due to arrest of development in intra-uterine life, of which the cause is unknown; or it may be secondary to cerebral inflammation, generally meningitic (Hammarberg), in which case, in addition to atrophy, there is also sclerosis of a much more pronounced character than is seen in primary atrophy. Hypertrophy of the cerebellum has been described occurring generally in cases of imbecility. Doursott has recorded eight such cases, in which the weight of the cerebellum and pons varied from 200 to 256 grammes.

Symptomatology of cerebellar disease.—Diseases of the cerebellum vary in their symptomatology with the time at which they are observed, the rate of their development, the position in which they are situated, and according as they affect the cerebellum alone or implicate directly or indirectly neighbouring centres, tracts, or nerves.

The most characteristic symptoms are an instability of station and locomotion, similar to that of alcoholic intoxication in different degree.

These symptoms are always seen in mammals immediately after the infliction of lesions on the cerebellum, as originally described by Flourens, but they tend to diminish with time. I may here quote the description which I have elsewhere (*Brain*, 1894) given of the effects of cerebellar lesions, as observed by Turner and myself:—

"When the cerebellum has been totally removed from monkeys—an operation which we have found to be a most formidable one, and more frequently fatal than successful—the animals exhibit the most tumultuous disorders of equilibrium, so that station and locomotion are, for the time, altogether impossible. Gradually, however, these tumultuous disorders subside to such an extent that the animal is able to sit up, with the help of some support to which it clings, and ultimately regains such a degree of stability that it may dispense with extraneous aid, but is so tottery that it falls over on the slightest disturbance or excitement. Its gait is of a peculiar sprawling character; at first, barely raising its abdominal surface from the ground, and planting its limbs wide apart so as to increase the basis of support; these being in progression raised and set down in a brusque and characteristically ungraduated fashion, which character the movements of the limbs retain for an indefinite period. Notwithstanding this extraordinary unsteadiness and instability of equilibrium, the animals are, however, able to grasp tenaciously with both hands and feet, so much so that it is sometimes more easy to drag the chair to which they may cling than loosen their grip; and they are able to climb a rope with agility hand-over-hand, in no way differing in this respect from perfectly normal animals. But the most noteworthy and persistent feature is the remarkable astasia or unsteadiness of the head, trunk, and limbs, which are either agitated by constant fine tremors apart from obvious muscular exertion, or exhibit oscillations of the typical disseminated sclerosis type on volitional exertion; so that, for instance, an attempt to lay hold of a piece of fruit excites such wild oscillations of the hand and arm that the object aimed at is either knocked away, or if seized, after many unsuccessful efforts, is with the utmost difficulty brought up and held to the mouth. This instability is a persistent feature, and though lessening to some extent, never entirely disappears, and was clearly evident in one of Luciani's monkeys a whole year after the operation.

"If only the lateral lobe, or one-half of the cerebellum, is removed, the persistent symptoms, after the cessation of the primary disorders, namely, the sprawling, ungraduated action of the limbs, and the tremors of instability on volitional effort, are confined to the same side as the lesion.

"If, on the other hand, the middle lobe is destroyed, or extensively injured, as by antero-posterior division, the symptoms are essentially of *the same character as those which follow destruction of the whole organ, but they do not affect one side more than the other, and, according to our observations, are more pronounced in the head and trunk than in* the limbs. They also appear to be less persistent, and, in the course of a few months, pass off to such an extent that, except on careful examination, it would be difficult to distinguish an animal so operated upon among its normal companions."

Let us now proceed to inquire how far these experimental results are in harmony with the facts of clinical observation.

The characteristic symptoms of cerebellar disease in man are a reeling, staggering, or drunken gait—the so-called titubation or cerebellar ataxy. The equilibrium is unsteady, and there is a continual tendency to stumble or fall over the most trifling obstacles, particularly on hurried movements or change of position. The patient, however, who is unable to stand or walk without assistance, is usually capable in the recumbent position of making the finest volitional movements with force and precision. There is no true incoördination, as all the muscles concerned in any particular act co-operate together in perfect harmony. They have none of the brusque, irregular, and sprawling character of locomotor ataxy. But, though these symptoms are pathognomonic of cerebellar disease, clinical records undoubtedly show that in a large number of cases of organic disease, or degeneration, of the cerebellum, they have not been observed.

In 64 cases of tumour and cyst of the cerebellum which I have examined, there was a more or less uncertain staggering or drunken gait in 46 (that is, 72 per cent); in 11 of these (17 per cent) the gait was so unsteady that the patient could not walk alone; in 13 (20 per cent) there was no affection of the gait.

Next as to the position of the tumour in these cases. In 28 of the 46 positive cases the middle lobe was directly affected; and not implicated, directly at least, in 17; though in 3 of them the middle lobe was stated to have been pressed upon. In a case, reported by Buzzard, in which the middle lobe of the cerebellum was far out of the reach of the growth, the gait was unsteady and staggering, and ultimately the patient became unable to stand. So in a case, reported by Friedeberg, of solitary tubercle in the outer and under side of the right hemisphere, and in a case under my own care, in which the tumour was situated in the region of the left flocculus and middle peduncle, the gait was very ataxic. Of the 13 negative cases, there was in several extensive destruction of one hemisphere (see cases by Booth, case 1, and Ogilvie). In only one of these was the middle lobe stated to have been directly implicated by the new growth, namely, a case by Leimbach. In this case the anterior part of the superior vermis and culmen monticuli were quite destroyed by a tubercle.

Of 16 cases of abscess which I have examined no mention is made of the gait in 10. It was normal in 1, namely, Handfield Jones's case; and it was of the usual reeling and staggering character in 5. In all these the abscess was situated in the lateral lobe. In a case of softening reported by Campbell (23), in which the uvula, pyramid, tonsil, velum medullare anterius, and the inferior surface of the whole of the left lateral lobe were destroyed to the depth of a quarter of an inch, there was no cerebellar ataxy.

In the reported cases of hæmorrhage the gait as a rule is not mentioned; but in Dana's second case the patient was sent to a police cell as intoxicated, there is no doubt, therefore, that he exhibited the characteristic symptoms.

Of cases of atrophy of the cerebellum, Nothnagel has referred to 13 in his well-known work. In 8 mention is made of uncertainty in the movements of the limbs, always of the legs, and sometimes of the arms; in all these the vermis was affected. In 5 there was no disturbance of co-ordination, but only 2 of these were available for deductions. In one of them, namely, Otto's case, the vermis was less affected by atrophy than the hemispheres; and in Lallement's case only the left lateral lobe was atrophic, while the right was unusually well developed, and the vermis appeared normal.

I have analysed 16 cases of cerebellar atrophy which have been reported since 1879, but some of them, owing to imperfect records, are not available for accurate deductions. And as the symptoms that have been observed in cases of atrophy are of especial value as indicating the results of cerebellar deficiency as such, apart from indirect influences, the principal facts of these cases are here appended in a tabular form :—

CEREBELLAR ATROPHY.—*Cases collected from* 1879 *to* 1895.

	Intelligence.	Speech.	Ataxy, Gait, etc.	Powers, etc.		P.-M.
1. CLAUS. *Arch. f. Psych.* xii. p. 682.	Very weak intellect. Epileptic.	Very defective and difficult.	Gait uncertain and staggering. Body bent forwards. Later, fell easily forwards. When he sat up, fell forwards.	*Once*, sexual excitement.	Left pupil dilated. Tongue went to left. Laughed almost only with right face.	Only normal parts of cerebellum were the inf. vermis and inner parts of both lower hemispheres. Claus thinks process began in pia.
2. KIRCHHOFF. *Arch. f. Psych.* xii. p. 647. Case 1.	Epilepsy from age of 5, mostly left-sided convulsions. Intelligence weak. Quite well up to age of 5.	Scanning and slow. Swallowing very difficult.	Drunken gait. Ataxy of arms, especially right. Ataxy of trunk and head on sitting up. Head and trunk bent forwards.	Motor weakness of legs unmistakable. No clear idea of position of limbs. Ordinary sensation intact.	. .	Left cerebellar hemisphere considerably smaller than right. Atrophy specially of posterior part of vermis and of left hemisphere. *Great hydrocephalus.*
3. Do. Case 2.	Weak intellect. Phthisis.	Slow and hesitating.	Movements slow and tremulous.	Strabismus convergens.	. .	Cerebellum very small and tough (that is, sclerosed). Almost complete atrophy of cerebellar cortex all over.
4. BOTELL. *Arch. f. Psych.* xv. p. 268.	Weak intellect. Irritable. Epilepsy from age of 10.	Slow and jerky.	Movements difficult and ataxic. Right side shoved forward. Swaying in standing. Phenomena less in excited state.	No paralysis.	Neck muscles in fixed tonic spasm.	Much atrophy of vermis and of both hemispheres, especially left (of left side only part of tonsil and flocculus left). Pons very small. No microscopic examination.

5. **Sommer.** *Arch. f. Psych.* xv. p. 252.	At 3 years old a severe brain inflammation. Weak intellect ever since.	Slow, drawling and monotonous.	No ataxy. Gait rather reeling and tended to fall forwards. Knee-jerks well retained.	Power and sensation normal. *No convulsions.* Great eroticism.	Gait attributed to hydrocephalus.	Great hydroceph. int. and ext. Partial atrophy of both hemispheres. Vermis intact.
6. **Major.** *Journ. of Ment. Sci.* 1882, 83.	Epileptic idiot from birth.	Not affected.	No ataxy. Gait not peculiar.	...	...	Atrophy of under surface of one hemisphere. Vermis and other hemisphere appear normal.
7. **Ferrier.** *Functions of the Brain*, 2nd ed., 1886.	Somewhat weak.	...	Could walk well and steadily, never ran.	General muscular weakness and tremor of hands—attributed to phthisical debility.	Senses normal.	Left cerebellar lobe a mere papilla, vermis a minute nodule. Right lobe constituted most of it, about ½ sq. inch and ¼ inch deep. Pons only a few transverse fibres. All peduncles very insignificant.
8. **Schultze.** *Virchow's Arch.* Heft cviii.	Weak intellect. Headache. Vomiting, giddiness.	Stammering.	Reeling gait.	...	A year later, symptoms of disseminated sclerosis, viz. nystagmus, intention-tremor, knee-jerks increased. Paræsthesiæ.	Atrophy and sclerosis of cerebellum and medulla. Primary degeneration in medulla.
9. **Ingels.** *Bull. de la soc. de méd. ment. de Belgique*, 1884, 35.	Weak intellect. Epileptic. Died in status epilepticus.	...	No ataxy. Quick and sure in every movement.	...	Like Otto's case, the hemispheres were more affected than vermis (Hammarberg).	Great atrophy mostly of hemispheres. Sup. vermis normal, inferior small. Pons reduced and like a band. No microscopic examination.

CEREBELLAR ATROPHY.—*Cases collected from* 1879 *to* 1895 (*continued*).

	Intelligence.	Speech.	Ataxy, Gait, etc.	Powers, etc.		P.-M.
10. HITZIG. *Arch. f. Psych.* xv. p. 267.	Slow intellect.	...	No ataxy. Could walk, spring, and dance.	.	...	Right cerebellar hemisphere very atrophic. Exact state of vermis does not seem to be known, not much seen of it. Right pons diminished.
..	..	Highly impaired.	Ataxy in all limbs, especially right. Reeling gait. Fell easily back.	Great muscular-weakness—legs weak. Right 7th nerve weak, also left. Left mydriasis, etc.		Left olive appeared wanting. No microscopic examination.
11. AMALDI. *Rev. Neurol.* 1894.	Melancholic..	..	. .	No motor or sensory disturbances.		Left cerebellar hemisphere reduced by half. Left Clarke's column, left posterior column nuclei, especially external part of Burdach's, right olivary body, right fibres of pons, right cerebral peduncle atrophied.
12. DERCUM. *Journ. of Nerv. and Ment. Dis.* 1893, p. 673.	Somewhat impaired.	...	Gait staggering. Difficulty in balancing. Tended to fall forwards or backwards.	Movements of hands very jerky and irregular. Knee-jerks diminished.	Partial optic atrophy.	Excessive atrophy and softening of cerebellum.

13. HAMMARBERG. *Nord. Med. Arkiv;* 1890, Bd. xxii. No. 23.	Normal up to age of 24, then chill and mental over-work led to depression. Paranoia. Fits of mania. Few and slight epileptic attacks. Lost power of orientation.	At first could not pronounce a single articulate sound. Incessant inarticulate sounds. Then speech came back indistinct and scanning.	At first continuous strong oscillations in limbs and trunk followed by ataxia, and gait remained staggering. Movements slow and uncertain. Speech and movement quite normal in maniacal excitement.	No loss of power. No tremors. No eye changes.	Patient was quite well up to age of 7, then cerebellar symptoms followed "a cerebral inflammation."	Atrophy and sclerosis of cerebellum. Microscopically vermis appeared normal, but microscopically no part normal; the part of vermis most affected being the post-lobules. Pia thickened all over cerebellar.
14. DOURSOUT. *Ann. med. Psych.* 1891, p. 345. 4th case of atrophy.	Mental debility.	Feeble, eunuch-like voice.	No affection of locomotion.	...	Great sexual excitement.	Weight of cerebellum about half the normal. No details.
15. BOND. *Journ. of Ment. Sci.* 1895, p. 409.	Since age of 7 mentally deficient. Dementia. No epilepsy. Maniacal outbursts.	Faltering, hesitating. Power of deglutition impaired.	Walked with difficulty. Ataxia of limbs. Much general tremor. Later, gait so ataxic that she could scarcely walk. Knee-jerks normal. Later left knee-jerk and right knee-jerk difficult to get.		Death at 60 from phthisis. No vomiting, vertigo, spasm of neck, nystagmus, or eye affections.	Most extensive and symmetrical atrophy and sclerosis of all parts of cerebellum. Cerebellum about $\frac{1}{3}$ normal weight. Pons about $\frac{1}{2}$ normal weight. Cerebrum appeared normal.
16. CRAMER.	Epilepsy since age of 14. Got worse. Epileptic mania and delusions.	Speech and swallowing difficult towards the end.	Tremors in limbs, especially left. In attacks head turned strongly to left, and eyes to left and down.	Motion and sensation normal. *After* an attack left arm paralysed.	Left upper eyelid lower than right. Pains in left face. Says she cannot see with left eye.	Right cerebral hemisphere about $\frac{1}{2}$ c.m. shorter than left. Left cerebellum about $\frac{1}{3}$ less than right. Vermis and right hemisphere appear intact.

An analysis of the facts of the different forms of cerebellar disease shows that the characteristic symptoms have been observed only in a certain proportion of the recorded cases. For the most part the negative cases have been those of slow growth or long standing, and where the disease was confined to one hemisphere. The characteristic disturbances are manifested most frequently when the middle lobe is affected. On this fact Nothnagel has founded the conclusion that the middle lobe alone exercises those functions which we ascribe to the cerebellum; and that on lesion of this lobe, directly or indirectly, the symptoms of cerebellar disease exclusively depend. This view receives no support from the phenomena of experimental lesions, as has been shown by Luciani, Turner and myself. Indeed, as has been mentioned above, the effects of destruction of the middle lobe are not more pronounced and are not more enduring than those which follow extirpation of the lateral lobe. A lesion of the middle lobe is more calculated to affect the organ as a whole than one situated in the lateral lobe; and the standard by which it is sought to determine that the middle lobe is really implicated in all cases in which the characteristic symptoms are manifested is a purely arbitrary one. The apparent discrepancy which exists between the many negative cases of cerebellar lesion in man and the uniformly positive results of experimental lesions in the lower animals is largely, if not entirely, explicable by the principle of compensation effected both by the other parts of the cerebellum and by the higher centres. This principle, which I enunciated many years ago, has been experimentally verified by the researches of Luciani. The disturbances of equilibrium are always most marked immediately after the infliction of the injury on the cerebellum. This may be accounted for by the suddenness of the derangement of the self-adjusting mechanism on which the maintenance of the equilibrium mainly depends. As, however, the loss of this mechanism may be to some extent compensated by conscious effort, the animal acquires in process of time the power of voluntary adaptation by means of its cerebral centres, and is thus enabled to maintain its equilibrium; though with a less degree of security than before. The more extensive the lesion, the greater the disturbance and the greater the difficulty of effecting by conscious effort all the muscular adjustments which are necessary to maintain the balance. Hence, even when the utmost degree of compensation has been achieved, a greater or less degree of instability and unsteadiness of movement and an incapacity for prolonged exertion are manifested. When the cerebellum is extensively diseased, or largely atrophied, all the motor adjustments concerned in equilibration, formerly easy and automatic, are now performed by the voluntary centres under a laborious sense of strained attention and conscious effort. No compensation takes place if, as Luciani has shown, the motor centres of the hemispheres are destroyed along with the cerebellum itself. Hence, congenital defect, or a slowly progressive lesion, affords the most favourable conditions for the supplementation of the higher centres of the automatic mechanism, which is originally deficient or gradually undergoing degeneration; and, as a rule, it has been found that congenital defect or extensive lesion of the cerebellum in early life has been associated with a tardy acquisition of station and locomotion; indeed it is questionable whether perfect compensation is attainable.

Rotatory movements.—It frequently happens that, after experimental lesion involving the lateral lobe and, more particularly, one of the cerebellar peduncles, forced or rotatory movements occur which tend to carry the animal round its vertebral axis. The direction of these movements has been variously described, and probably they are not altogether uniform, but vary as the lesion has more of an irritative or a destructive character. In my own and Turner's experiments, which correspond with those of Magendie and Hitzig, the almost invariable direction, while the animal was sprawling on the abdominal surface, was a tendency to roll, or an actual and apparently irresistible rolling towards the side of lesion. Thus, if the left lateral lobe were destroyed, or the middle peduncle divided, the animal, laid on its ventral surface, would roll to the left. This rolling tendency may be described as the effect of a rotation round the vertebral axis from left to right; that is, from the injured to the sound side. Russell (94), however, represents the rotation as occurring in the inverse sense.

Rotatory movements have not often been observed in man. They seem to occur more particularly when the lesion involves the cerebellar peduncles. Not infrequently, however, there are forced movements (Zwangsbewegungen), which are of a similar character to the rotatory or rolling movements seen in the lower animals, and cause the patient to assume positions from which he cannot be easily displaced, or to which, if left alone, he tends to return. The direction of these movements is, however, not uniform. Jalland reports a case of tumour of the right side of the cerebellum compressing the medulla, in which the patient always lay more on the right side than on the left. Two days before death the patient turned on the left side, and if laid on the right side would turn over on the left. Wulff (105) has recorded a case of tumour affecting principally the left side, in which the patient ultimately lay continuously on the right side; and Friedeberg (case 9) reports a case of abscess of the left hemisphere in which the forced movements were bending of the body forwards and of the head backwards.

Tremors.—Mention has already been made of a remarkable astasia or unsteadiness of the head, trunk, and limbs in monkeys after the primary tumultuous disorders of equilibrium have passed away. If only one lateral lobe has been destroyed, this astasia occurs specially in the limbs on the side of lesion. There is either a constant fine tremor apart from obvious muscular exertion, or oscillation of the disseminated sclerosis type on volitional movement. These tremors we have found to be most marked in the upper limb, and they tend to subside in course of time.

The number of cases in which tremors have been recorded in man is relatively small, compared with those in which the other characteristic symptoms have been observed. In 100 cases of cerebellar disease, referred to by Krauss, tremors were only noted in two or three. In Wetzel's collection tremors of the hands were observed twice, and of the limbs once. Among those analysed by myself, intention-tremors of the hands or arms were observed in three cases. In one of these, reported by Suckling, there was intention-tremor of the right arm; in this case the whole of the left hemisphere, with part of the vermis, was affected by tumour. In Ackermann's case there was characteristic tremor of the arms and legs; in this case the whole of the middle lobe, as well as each hemisphere, was affected. In ten cases of tumour that have come under my own observation tremors were noted in six. In one case (tumour of the right cerebellar hemisphere) there were fine tremors of the head, neck, shoulders, and arms, especially the left. In a second case (tumour of the left lobe pressing on the middle lobe) there were slight volitional tremors in both hands. In a third case (tumour invading the left hemisphere and superior crus cerebelli) there were tremors of the left arm when extended. In a fourth (tuberculous mass in each cerebellar lobe) there were tremors, especially of the right hand; and in a fifth case (tumour pressing on the left cerebellar hemisphere from above) tremors were observed in the right hand and foot. In a sixth case (tumour affecting the left side of the cerebellum and pons) there were tremors of both hands, but especially of the right.

Tremors or tremulous movements have been observed in a considerable number of the cases of atrophy of the cerebellum, even when the pathognomonic instability of equilibrium has not been specially noted; particularly those of Kirchhoff (case 2), Shuttleworth, Dercum, and Bond (table, pp. 366–369).

The results of experiments upon monkeys would lead one to expect that in cases of cerebellar disease in man the intention-tremors would have been more frequently observed than actually appears to be the case; also that the tremors would have been more especially observable on the side corresponding to the lesion. But in this respect the recorded facts are so discrepant that little reliance can be placed upon them as a means of precise diagnosis of the seat of lesion. This illustrates the great difficulty of arriving by clinical investigation alone at the real cause of the functional disturbances met with in cerebellar disease. There can be little doubt that the cerebellar innervation of the limbs is direct and not crossed; but it is difficult to find an exact formula to express the nature of this innervation, or to give an anatomical or physiological explanation of the manner in which the cerebellum exercises its influence on the muscles. Luciani (64) is of opinion that the cerebellum normally exerts on the apparatus of movement a *sthenic*, *tonic* and *static* influence, so that destructive lesion or defect of the organ exhibits itself in a condition of *asthenia*, *atonia* and *astasia*, which he regards as the essential condition of cerebellar ataxy. Turner and I, after experimental lesions of the cerebellum of monkeys (39), have failed to discover any satisfactory evidence of asthenia, or atony of the muscles. On the contrary, even in animals which were absolutely unable to maintain their equilibrium, we have observed such indications of muscular vigour in the limbs, that the term asthenia seems to us a misnomer. Furthermore, clinical observation has abundantly shown that in cases of atrophy or other disease of the cerebellum, such as to render station and locomotion altogether impossible, patients in the recumbent position may perform all movements of the limbs with energy and precision. Luciani's term astasia, however, very aptly describes the primary instability both of station and locomotion, as well as the subsequent and more persistent tremor and unsteadiness, not only of the body as a whole but of the limbs also, which are so uniformly observed after experimental lesions in monkeys and dogs. The hypothesis which appears to me best to explain this astasia, or unsteadiness, is that propounded by Dr. Hughlings Jackson, according to which "all the muscles of the body are innervated both by the cerebrum and cerebellum, but in an inverse order. The cerebellum regulates the muscular contractions necessary for our attitudes in space, while the cerebrum regulates the contractions necessary to effect all changes of attitude which are made in response to successive impressions occurring in time. . . . Speaking broadly, then, the cerebellum regulates continuous or tonic muscular contractions. It will be seen, therefore, that every combined muscular adjustment necessitates the co-operation of both these organs; no change of attitude can be effected by the cerebrum, except in so far as a certain attitude was previously maintained by the cerebellum, and no steady movements can be produced by the alternate contractions of some groups of muscles, except in so far as other groups of muscles are maintained in a state of continuous contraction. Hence it may be inferred that all movements of the body are co-ordinated both in the cerebellum and cerebrum" (90*a*). The cessation of the tonic or continuous innervation of the cerebellum should, according to this hypothesis, exhibit itself in an unsteady and ungraduated character of all the motor adjustments, whether of the body as a whole or of its individual parts; and should theoretically induce a state of affairs which actually agrees well with the effects of cerebellar lesions in animals.

Palsies.—It is unquestionably the rule, both in experimental lesions and in the phenomena of cerebellar disease (as such) in man, to find an entire absence of paralysis or paresis properly so-called. Luciani carefully distinguishes between what he terms asthenia and paralysis, though Russell (93) seems to ignore the distinction. Yet there are many cases of cerebellar disease on record in which a paralytic or paretic condition of the limbs has been observed, sometimes on the side of lesion, at other times on the opposite side; in most cases of cerebellar atrophy, however, apart from general weakness, unsteadiness, or tremors, there is no true paralysis or paresis. In cats, in which this condition seems to be not uncommon, the symptoms have been variously described. In one case, reported by Rumpf, there were no paralytic symptoms; in another, recorded by Krohn, there was at first paralysis of the hind legs, which, however, resolved itself into mere clumsiness of the limbs; and in a third, described by Carruthers, in which the right hemisphere was much smaller than the left, there was paresis on the right side and slight weakness of the left leg.

Among cases of softening, one has been reported by Atkins of complete right hemiplegia and hemianæsthesia, in which the left lobe was broken down; but in this case the posterior end of the left optic thalamus did not appear healthy. In a case, reported by Menzies, of thrombosis of the left posterior inferior cerebellar artery there was weakness of the limbs and the face on the left side; and in another by Campbell (23), of softening of the left side, there was temporary complete hemiplegia, followed by permanent paresis of the left leg. The same author (24) also records a case of softening of the vermis and both hemispheres, especially the left, in which there was relative weakness in the left arm and leg.

In 16 cases of abscess paralysis is said to have existed in 6, and is not mentioned in 10. The limbs were affected on the same side as the lesion in 3 cases; namely, the arm, in a case reported by Macewen; the arm and leg in one (case 3) by Krauss; the arm paralysed, and leg and face weak, in one by Drummond.

Of 9 cases of hæmorrhage, paralysis was observed in 5. It was on the side opposite the lesion in Farquharson's case; on the same side in a case reported by Thacker. There was gradually increasing weakness of the arm on the side of lesion in a case reported by Friedeberg; and in one by Dana the limbs on the side of lesion offered less resistance to movement than those on the opposite.

Of 64 cases of tumour or cyst there was no paralysis in 21. There was weakness of the limbs on the side opposite the lesion in 2 cases reported by Wetzel and Suckling respectively. There was weakness of the opposite limbs, and also of the trunk muscles, in a case, reported by Jackson and Russell, of a cyst occupying chiefly the inferior vermis and involving the lateral lobes, especially the left. In this case the medulla was flattened and the lateral ventricles distended, causing also compression of the cerebral convolutions. The epposite arm was said to be weak in a case recorded by M'Burney and Starr. In this case the tumour occupied the left hemisphere, and caused obvious pressure on the left half of the pons. In a case reported by van Hell of tumour of the antero-superior portion of the left hemisphere and vermis, there was paresis of the face and arm on the opposite side. There was weakness of the limbs on the same side in cases reported by Luderitz and by Krauss (case 2); and in five others the arm alone was said to have been weak on the side of lesion. Weakness of both legs has been observed in cases of tumour of one side, as in a case reported by Buzzard, and in another under my own care; and other cases have been recorded in which there was weakness of the limbs on the side of lesion and of the leg on the opposite (*vide* cases by Booth, Friedeberg (case 3), and Dreschfeld). In Dreschfeld's case there was a tumour of the left cerebellar hemisphere with left hemiparesis and weakness of both legs. In a case, under my own care, of tumour of the middle lobe there was gradually increasing weakness of the left arm and leg. In Wetzel's collection of nine cases of tumour of one hemisphere, paresis was observed four times on the side of lesion, four times on the opposite, and once on both sides. Double-sided weakness, especially of the lower limbs, occurs, according to him, in tumours of the vermis, and when there is distension of the lateral ventricles.

It appears, therefore, from a consideration of the above data, that when paralysis or paresis occurs in connection with cerebellar disease there is no rule as to its occurrence on the one side or the other. It is obviously an indirect effect, sometimes due to direct pressure on the motor tracts above the decussation, or to counter-pressure of the same against the edge of the tentorium and petrous portion of the temporal bone; while at other times it is difficult to give a satisfactory explanation from the recorded data alone. The simple fact that the characteristic phenomena of cerebellar disease may occur in the entire absence of paralytic or paretic symptoms is sufficient to prove that these symptoms, when they do occur, must be attributed to some other cause than to the cerebellar affection, as such.

Convulsions.—Allied to the symptoms which we have been just considering, but of an inverse order, are those of rigidity or spasm in connection with cerebellar lesions. That irritation of the cerebellum may cause muscular spasms of the limbs on the same side of the body I have myself shown by the direct application of the faradic current to the cerebellar cortex. Forced movements of a peculiar character, which are regarded by Luciani as being due to irritation, are seen in animals after removal of one cerebellar hemisphere; but more especially, as Turner and I have found, after division of the peduncles, especially the inferior. In the latter case there is in monkeys pleurotonus to the side of lesions, with adduction and flexion of the limbs on this side, and abduction and extension of the limbs on the opposite. In dogs, according to Luciani, instead of flexion of the fore limbs there is extension. The spasm, however, is not of a rigid character, and can easily be overcome. Though this forced position is regarded by Luciani as due to direct irritation, yet, inasmuch as it occurs immediately after the lesion and before inflammatory irritation can have been set up, it is probably only a dynamic effect, and due to the suddenness of the disturbance.

General convulsions in connection with cerebellar disease are probably of an indirect character. It is possible, however, that unilateral rigidity of the limbs on the side of lesion may be due to direct irritation of the cerebellum itself. In a case of disease of the left lobe, reported by Macewen, there was rigidity with flexion of the left arm, and some rigidity of the muscles of the neck and jaws. In a case of abscess of the right lobe, reported by Drummond, there were right-sided convulsions, beginning in the face, and spreading to the arm, and occasionally to the leg. Convulsions and retraction of the head, with rigidity of the masseters, have been observed in other cases of abscess, and in fact are so common that they are regarded by Friedeberg as pathognomonic. These symptoms, however, most commonly occur when inflammation has been set up about the pons and medulla.

Tonic rigidity of the muscles of the back of the neck, with retraction of the head, associated with flexion of the forearms and extension of the legs with pointing of the toes, has been described by Hughlings Jackson as an especial feature of tumours of the middle lobe. He regards these spasms as the direct result of cerebellar irritation, but this is open to question; in my opinion they are more probably indirect effects.

The knee-jerks.—There is great variation in the condition of the knee-jerks in cerebellar disease. After experimental ablation of the cerebellum in monkeys, Turner and I have found that the knee-jerks gradually become increased, though at first there is no obvious alteration.

After extirpation of a lateral lobe the tendency is for the knee-jerk on the side of lesion to become increased, if this do not occur immediately after lesion. Russell (94) finds in rabbits and dogs that immediately after unilateral extirpation the knee-jerk is increased on the side of lesion, and diminished on the opposite side. Later the knee-jerk on the side of lesion becomes less active, until they become nearly equal. The relation of the cerebellum to the knee-jerks has been the subject of some speculation, but nothing has as yet been satisfactorily determined. Hughlings Jackson and Bastian are of opinion that the occurrence of rigidity, with increased knee-jerks, in limbs paralysed from cerebral disease, is due to the "unantagonised" or "unrestrained" influence exerted by the cerebellum. When in man the limbs are paralysed by total transverse lesion of the spinal cord, the knee-jerks are absent, and the limbs do not become rigid. This result is regarded by Bastian as due to the cutting off of the cerebellar influence, hypothetically exerted through the gray matter of the cord. It is not the case, however, as we have seen in animals, that the destruction of the cerebellum annihilates the knee-jerk; on the contrary the knee-jerks actually become increased, and in the lower animals, even in monkeys, as Turner and I have shown, total transverse section of the spinal cord does not prevent the occurrence of rigidity with increased knee-jerks. The mechanism of this condition must therefore exist in the spinal cord itself, and cannot be regarded as dependent on the cerebellum. Krauss finds that in 100 cases of cerebellar disease the knee-jerk is mentioned as being normal in 10; increased in 12; diminished or absent in 12. Oppenheim states he has never seen a case of uncomplicated cerebellar disease in which the knee-jerks were absent. In one case in which this occurred there was also tabetic degeneration; and this was likewise present in a similar case reported by Wollenburg. In the recorded cases of cerebellar atrophy generally no mention is made of the state of the knee-jerks. They were well-marked in a case reported by Somer; at first normal, and later unequal in a case reported by Bond, and in Dercum's case diminished.

Wetzel in his collection of 59 cases of cerebellar tumour finds the knee-jerks generally well marked; they were increased in 3, and diminished in 1 case. Among 19 cases of abscess the knee-jerks were increased in 2, diminished in 2, normal in 4, and not mentioned in 11. Among 64 cases of tumour or cyst the knee-jerks were increased in 15, diminished or absent in 14, and normal in 10. Of the cases in which the knee-jerks were increased the disease was situated on one side in 8, in the middle lobe and hemisphere in 3, and in the middle lobe in 1. Of those in which the knee-jerks were diminished or absent the lesion was in one hemisphere in 8 cases, and in the middle lobe in 4. In those cases in which the knee-jerks were normal, the lesion was in one hemisphere in 4, in the middle lobe and one hemisphere in 3, and in the inferior vermis in 1.

It appears, therefore, from this analysis that in lesions of almost any extent or position the knee-jerks may be increased, diminished, or normal. The state of the knee-jerks may also vary at different times in the same case. Thus in a case, under my own care, of carcinoma of the left side of the cerebellum, the knee-jerks were at first equal and normal; at a later date they were somewhat increased, and still later very slight. In another case in which there was a cyst of the left lateral lobe excavating the middle lobe and portion of the right lobe, the knee-jerks were equal and normal; at a later date they were diminished, especially the left; still later both were active and equal, and ultimately the left was almost gone, and there was a tendency to ankle clonus.

Affections of general and special sensation.—Most experimenters are agreed that lesions of the cerebellum cause no perceptible impairment of cutaneous sensibility; and this is in general confirmed by the results of clinical observations in man. In some cases, however, some degree of hemianæsthesia has been observed on the same, or on the opposite side, just as in analogous cases of hemiparesis. As a rule, in cases of atrophy of the cerebellum no mention is made of affections of sensation; and the same is true of all the cases of hæmorrhage and abscess to which I have been able to refer. In Atkins' case of softening, in which, however, there was probably some implication of the optic thalamus, there was hemiplegia with hemianæsthesia on the opposite side. In Wetzel's collection of 59 cases of cerebellar tumour sensory impairment is not mentioned once. In my own collection of 64 cases of cerebellar tumour, or cyst, sensation was especially mentioned as being intact in 32, and not mentioned at all in 25. In a case, reported by Jalland, of tumour on the right side, there was some difficulty in localising tactile impression on the arm on the side of the lesion. In a case, mentioned by Booth, of myxo-sarcoma of the left hemisphere, the tips of the fingers of the left hand were slightly anæsthetic; and in one, observed by Knapp (case 28), of tumour on the under surface of the cerebellum implicating both lateral lobes, the left more than the right, sensation on the right hand was somewhat diminished as compared with the left. It is evident from these facts that it is only in cases of disease of such a nature as to produce indeterminate indirect effects that sensory disturbances are observed.

The phenomena of so-called cerebellar ataxy have been ascribed by Lussana to loss of muscular sense; but the experiments of Luciani, as well as those of Turner and myself, lend no support to this surmise. When it occurs in cerebellar disease it must be regarded purely as an indirect symptom, and not due to lesion of the cerebellum as such. In some cases (Preston, 89) muscular sense is said to have been impaired or lost, apart from any affection of general sensibility. On this point, however, further investigation is necessary.

Vision.—Experimenters have generally failed to obtain any evidence of affection of the special senses after the most extensive lesions or total extirpation of the cerebellum. In those cases of cerebellar disease, such as atrophy, in which there is no indirect affection of the optic nerve or tracts, by pressure or neuritis, there is no impairment of vision. Some forms of cerebellar disease, however, especially tumour, lead very commonly to impairment of vision, more so perhaps than tumours situated elsewhere. This is probably due to the greater frequency and intensity of optic neuritis combined with hydrocephalus.

Optic neuritis occurs as a rule very early in the course of cerebellar tumours and often leads to blindness. Neuritis, however, may be entirely absent in cases of cerebellar tumour, even when it has reached a considerable size. In Bernhardt's collection of intracranial tumours impairment of vision occurred in 61 per cent of cerebellar tumours, and in 36 per cent of cerebral tumours. In Wetzel's collection of cerebellar tumours there was impairment of vision in 81 per cent, and total blindness in 11 cases. Of 64 cases of cerebellar tumour which I have analysed, optic neuritis was present in 46, absent in 4, and not mentioned in 14. It was present in 9 out of 10 cases under my observation. Of these 64 cases there was impairment of vision in 35, and total blindness in 21. Among the 35 cases optic neuritis was especially noted in 31, and was not mentioned in 4; therefore there was no case of failure of vision where optic neuritis was expressly excluded. Among the 28 cases in which the state of vision was not specially mentioned, 15 had optic neuritis, 4 had no neuritis, and in 9 no mention was made on the point. In some, vision failed first on the side of lesion, and in others on the opposite.

Affections of hearing.—Affection of hearing not infrequently occurs in cerebellar disease, but only under conditions calculated to press upon the auditory nerves or tracts. Bernhardt found affection of hearing, almost always unilateral, in 18 per cent of tumours of the vermis; in 26 per cent of tumours of the hemispheres. Among my own 64 cases of tumour hearing was affected in 19. In 11 it was on the side of lesion only; and in 4, more on this side than the other. In 2 cases hearing was impaired on both sides; and in 2 others there was complete deafness on both sides. In connection with tumours of the middle lobe noises in the ears were noted in 4; and hallucinations of hearing were observed in a case reported by Luderitz. It is not improbable, as Dercum suggests, that the deafness present in some cases of cerebellar tumour may be due to pressure on the posterior tubercles of the corpora quadrigemina and internal geniculate bodies, through which the central auditory tracts appear to ascend.

Affections of smell and taste.—In pure uncomplicated cases of destructive lesion of the cerebellum affections of smell and taste are unknown. In cases of tumour, however, no doubt owing to the pressure, they are sometimes met with. We find loss of taste mentioned in five cases. It was lost on the side of lesion in a case reported by Wetzel; in another by M'Weeney; and defective in a case reported by Buzzard. In a fourth case, under my own care, of a cyst of the size of a turkey's egg in the left cerebellar hemisphere excavating the middle lobe and part of the right lateral lobe, taste and smell became defective on the left side, and ultimately smell was lost in both nostrils; and in a fifth, of cyst of the posterior part of the middle lobe, taste was defective on the right side. I find loss of smell mentioned in 5 cases, namely, in cases reported by Wollenberg, Knapp, and in 3 others under my own care. Smell was defective on the side of lesion in the case reported by Jackson and Russell.

Affections of the ocular muscles.—I have found that electrical irritation of the cerebellum in different animals causes ocular movements varying with the position of the electrodes on the different lobes; and one of the remarkable symptoms, described by Magendie as resulting from division of the middle peduncle, was a skew deviation of the optic axes—the eye on the side of lesion being directed downwards and inwards, the other upwards and outwards. Luciani has also obtained similar results on removal of one-half of the cerebellum, and looks upon them as the result of irritation. Russell (93) believes that the ocular deviations in cerebellar disease are paralytic, and that the eyes in uncomplicated unilateral lesions turn from the injured side. Turner and I found the ocular deviations following cerebellar lesions in monkeys so variable that we could trace no constant relation between them and the seat of the lesion.

In cases of cerebellar atrophy ocular deviations, or paralyses, are practically unknown, though a case of convergent strabismus is mentioned by Kirchhoff. In tumours, however, and such forms of disease as cause pressure effects, ocular palsy is not unknown. Bernhardt records paralysis of the sixth in 3 cases; and Wetzel in 8, namely, 4 times in tumour of the hemisphere, 3 times in tumour of the middle lobe, and once bilateral in a case where the exact situation of the tumour was doubtful. Among my 64 cases of tumours and cysts ocular palsy was mentioned in 24. The external rectus was paralysed on the side of lesion in 6 cases of tumour and 2 cases of abscess; it was paralysed on both sides in 7 cases. Diplopia was mentioned in 2 cases, and convergent strabismus in 1. These cases were in all probability due to paralysis of the sixth. In a case reported by Suckling, conjugate movement of the eyes was lost to the side opposite the lesion, and was weak on the same side. Conjugate deviation of the eyes to the opposite side also occurred in 2 of the cases collected by Wetzel, and in 3 of those by Bernhardt. In a case reported by Eskridge, there was convergence of the eye on the side of the lesion, and divergence of the eye on the opposite side. The patient could not turn the eyes down, and only very feebly upwards. The upward and downward movements of the eyeballs were weak in a case reported by Bohm. The eye on the side of lesion deviated outwards in a case reported by M'Burney and Starr. Both eyes were directed towards the side of lesion in a case reported by van Hell, and more or less complete ophthalmoplegia has been noted in 4 other cases. Ptosis has also been observed in several instances, sometimes on the side of lesion and sometimes on the opposite. The fourth nerve is rarely affected; it is mentioned as having occurred once by Bernhardt.

The ocular paralyses above described are without doubt due to pressure on the oculo-motor nerves—third and sixth, or on the corpora quadrigemina. We have seen that tumours of the corpora quadrigemina are more frequently accompanied by ophthalmoplegic symptoms, and therefore the absence of such symptoms, with unsteadiness of equilibrium and reeling gait, would be in favour of the situation of lesion in the cerebellum.

In addition to the deviations of the optic axes and paralyses of the ocular muscles, nystagmus not infrequently occurs. I have observed this phenomenon after electrical irritation of the cerebellum in animals, and it is commonly observed, temporarily at least, after experimental lesions, and constitutes one of Luciani's phenomena of irritation. Russell (94) is of opinion that in lesions of the lateral lobe the nystagmus occurs towards the side of lesion, while in lesions of the middle lobe the nystagmus is irregular or of a rotatory character. Turner and I have more frequently observed nystagmus in connection with lesions of the peduncles.

Nystagmus has not been noted in cerebellar atrophy. In 19 cases of abscess it is mentioned as having occurred twice. In 31 cases of tumour it was present in 19. In 10 of the cases under my own observation it was present in 5. Bernhardt mentions it as having occurred in 4 of the cases in his collection, and Wetzel in 5.

Exophthalmos has been observed in 3 cases of tumour; namely, van Hell, Booth (case 1), and Friedeberg (case 1).

The pupils.—The state of the pupils varies considerably in cerebellar disease. Among Wetzel's cases of tumour no reaction to light occurred in 7. In my own collection the pupils were dilated and fixed in 8, in all of which there was blindness; and dilated and sluggish to light in 8. They were dilated in 3 cases of tumour and 2 of abscess, and not contractile to light in 3 cases. The pupils were contracted in 3 cases of tumour, and contracted and fixed in 2 cases of hæmorrhage, reported by Mills and Oliver. They were first dilated and then contracted in a case of tumour reported by van Hell; and first contracted, and then dilated before death, in a case of hæmorrhage reported by Barling.

The pupils are frequently unequal: thus the pupil was larger on the side of lesion in 5 cases of tumour and 1 case of hæmorrhage; and larger on the opposite side in 6 cases of tumour and 1 of abscess. In a case under my own care of tumour of the left lobe, the left pupil was at first larger than the right, and at a later date the right was larger than the left.

It is evident, therefore, from these facts that no reliance can be placed upon the condition of the pupils as a means of regional diagnosis of the seat of lesion.

Affections of other cranial nerves.—In addition to the affections of the cranial nerves above mentioned,—namely, the first, second, third, fourth (rarely), sixth (often), and eighth (occasionally), other cranial nerves may suffer indirectly from cerebellar lesion.

The fifth cranial nerve not infrequently suffers, either the sensory or the motor division or both. In the first case we have anæsthesia or paræsthesia in some of the regions supplied by the fifth. There was paræsthesia of the face on the side of lesion in a case reported by Wetzel; in a second by M'Burney and Starr; in one (case 28) reported by Knapp, and in another by this author (case 29), there was numbness of the face, which was also somewhat swollen on the side of lesion. In a case under my own care there was facial neuralgia on the opposite side. Occasionally there is anæsthesia of the cornea on the side of lesion, and this may be accompanied by neuro-paralytic ophthalmia. There was anæsthesia of the cornea in cases reported by Preston (case 3), Jalland, and Wollenberg. In the last-mentioned case the anæsthesia was total on the side of lesion, and almost total on the opposite side; in a case under my own care of tumour in the region of the left flocculus there was anæsthesia of both corneæ. In this case there was almost complete paralysis of the muscles of mastication on the left side.

Paresis of the muscles of mastication has also been reported in two of Wetzel's cases; and rigidity of the masseters is frequent, according to Macewen, in advanced stages of abscess. The affections of taste which have been noted above (p. 379) are no doubt due to indirect pressure on the fifth nerve.

Affections of the face or seventh cranial nerve.—Affections of the face are very common in cerebellar disease, and are almost always unilateral. In my collection of 64 cases of tumour I find facial paralysis mentioned as occurring in 21. In 3 of these there was weakness of the face on the side opposite the lesion. In Wetzel's there was weakness of the opposite side of the face and corresponding limbs. In van Hell's case the same condition occurred with doubtful weakness of the corresponding arm; while in Friedeberg's (case 2) the limbs were unaffected.

In 16 cases the facial paralysis was on the side of lesion. In Suckling's case there was paralysis of the face on the side of lesion, and of the limbs on the opposite. In Jalland's case there was weakness of the palate as well as of the face on the side of lesion; and a similar condition existed in a case reported by Purves Stewart. In a case under my own care both the upper and lower facial regions were *affected. In 2 cases of tumour of the middle lobe there was weakness of the face on one side. Among my cases of abscess of the cerebellum, weakness of the face is mentioned as having occurred in 2; namely, face and limbs on the same side (Drummond), and of the face on the side opposite the larger abscess in a case of bilateral abscess reported by* Friedeberg.

In a case of left-sided softening reported by Menzies there was facial paralysis on the same side; and in one of hæmorrhage recorded by Friedeberg there was paralysis of the face on the opposite side.

Affections of the tongue or hypoglossal.—Affections of the tongue or hypoglossal nerve are also not infrequent. Among my 64 cases of tumour it occurred 13 times—11 times on the side of lesion, and twice on the opposite. It is for the most part slight and often doubtful. Articulation is often impaired. It is difficult, hesitating, indistinct, slow, and sometimes of a scanning character. This symptom was present in 17 per cent of Bernhardt's collection of cerebellar tumours, in 10 per cent of Wetzel's, and in 15 per cent of my own. By most authors the impairment of articulation met with in cases of tumour is regarded as due to pressure on the pons or medulla; but it has been met with also in cases of cerebellar atrophy, and thus it becomes a question whether the affection of articulation may not be of a similar character to the ataxy or astasia of the limbs. Nothnagel found impairment of articulation in all (namely, 8) cases of total cerebellar atrophy, and absent in 4 out of 5 cases in which the middle lobe was not affected; and the symptoms in a case reported by Hammarberg are strongly in favour of the articulatory disturbances being the direct result of cerebellar lesion. In this case the symptoms came on at the age of seven after a so-called inflammation of the brain, and for three months there were continuous strong oscillations of the limbs and trunk, the patient being unable to pronounce a single word, and incessantly emitting inarticulate sounds. The oscillations gradually became less marked, and at the same time the power of articulation improved, but remained always indistinct and scanning. There was well-marked atrophy with sclerosis of the cerebellum, but no degenerative changes could be found in the tracts proceeding from the articulatory centres of the cortex.

Difficulty of swallowing is also met with occasionally, especially in tumours. It was present in 6 of Bernhardt's cases, 3 of Wetzel's, and 11 of my own. It has also been observed in some cases of atrophy (see cases by Kirchhoff (case 1), Bond, Cramer).

The pulse is occasionally affected, being sometimes quickened and sometimes slowed, more particularly in cases of tumour, abscess, or hæmorrhage. The same is true also of respiration. These disturbances of the pulse and respiration are no doubt only indirect effects.

Trophic influence of the cerebellum.—Luciani regards the cerebellum as exerting a trophic influence on the tissues—a supposition which he bases on the occurrence of certain cutaneous and other dystrophies after extirpation of the cerebellum in animals. Among these effects he describes polyuria, glycosuria, acetonuria, marasmus, cutaneous affections, conjunctivitis, etc. Turner and I have not been able to confirm Luciani's views of the influence of the cerebellum on nutrition; nor do they receive much support, if any, from clinical observation. Polyuria or glycosuria has occasionally been met with in cases of tumour, and Oliver records a case of hæmorrhage into the cerebellum in which glycosuria occurred, which did not exist before the lesion. The probability is that the glycosuria in this instance, as in the others, is due to irritation of the floor of the fourth ventricle, in accordance with Claude Bernard's experiments.

Mental changes.—Some authors (Nothnagel, Gowers, Kirchhoff) are of opinion that the cerebellar hemispheres are in some way related to the psychical functions; but the results of experimental physiology lend no support to this hypothesis. It is no doubt true that in most cases of cerebellar atrophy there is some impairment of intelligence, varying from mere weakness of intellect up to imbecility or even idiocy; but this is more probably the result of some cerebral degeneration coincident with that which had led to the cerebellar atrophy. This view is borne out by the facts of Hammarberg's case, in which the cerebellar symptoms began at the age of seven; yet there was no disturbance of intelligence until the age of twenty-four, and that after a chill and mental overwork. In tumours of the cerebellum mental hebetude or stupor is not uncommon, but this may with good reason be ascribed to secondary hydrocephalus.

Mental dulness and apathy were present in 33 per cent of Bernhardt's cases, in 35 per cent of Wetzel's, and in 37 per cent of my own. At other times the mental condition is one of irritability and restlessness. Occasionally there are hallucinations of sight or hearing.

Influence of cerebellar lesions on life.—Cerebellar disease, as such, is compatible with long duration of life. Thus in a case, reported by Bond, of complete sclerosis of the cerebellum, the duration of life was fifty-three years, death occurring not from the cerebellar disease, but from phthisis. In those forms of disease such as tumours, calculated to produce indirect disturbance of other centres, the duration of life varies. Cerebellar tumours often run a very rapid course. The average duration is two years (Preston). It is a noteworthy fact, however, that, after several months of active disturbance, periods of remission may occur, extending for several years, during which the patient may continue apparently well, with the exception, perhaps, of impairment or even total loss of vision dependent on post-neuritic atrophy. Death occurs not infrequently quite suddenly, apparently from sudden failure of respiration. This mode of death occurs in from 20 per cent to 25 per cent of cases of tumour.

Accessory symptoms.—Certain other symptoms are met with in cerebellar disease, which, though not of themselves of a localising value, are of some importance taken in conjunction with the other direct and indirect symptoms described above. These are vertigo, vomiting, and headache. Vertigo, though not characteristic of cerebellar disease, is specially frequent and often very distressing. Nothnagel is inclined to regard it as a direct symptom, but this is more than doubtful, as in the great majority of the cases of atrophy it is absent. It is present, however, in a large proportion of the cases of tumour, and occurs frequently also in cases of abscess, softening, and hæmorrhages. The same may be said of vomiting. It is more frequent and more persistent than in similar disease affecting the other intracranial centres. It is absent in cases of atrophy, and is more particularly associated with those forms of disease which encroach on the posterior fossa of the skull. The special frequency of vomiting under such conditions may perhaps be due to affection of the medulla oblongata and roots of the vagus.

The headache of cerebellar tumour is an early, and often for a long time the only symptom. It is often very intense. The position of the headache does not necessarily correspond to the situation of the disease. Though often occipital, it may be wholly or mainly frontal, or temporal. In one case of long duration, under my own care, pain was frontal for eighteen years, and occipital during the last four years. A peculiar case has been reported by Purves Stewart. This was a cystic tumour of the right cerebellar hemisphere, with persistent boring headache, strictly localised to a small area in the left frontal region. He is of opinion that the situation of the pain in this region may be accounted for by the anatomical connection of the cerebellar hemisphere with the opposite frontal region of the cerebrum, and that a pain of this character may therefore have localising value. Knapp also records a case in which there was such marked tenderness over the opposite temple that the patient was trephined in this region.

My own experience has led me to attach comparatively little importance to the situation of the pain spontaneously complained of in intracranial disease. It is otherwise, however, with the pain which is elicited by deep pressure or percussion. In a large proportion of the cases of cerebellar disease, tenderness or decided pain may be elicited in the occiput, even though not spontaneously complained of; and, according to my own observation, it is most marked on the side corresponding to the disease; though in some cases the whole occiput is so tender that it is difficult to be certain of the predominance of one side over the other. In the absence of other localising symptoms I attach great importance to the seat of the greatest intensity of the pain on pressure as an index of the probable position of the lesion.

Heredo-ataxie cérébelleuse (cerebellar heredo-ataxy).—Under the above title Marie (73) first described this affection, which he considered dependent on atrophy of the cerebellum; he founded his nosological classification upon certain cases observed by himself, together with others reported by Fraser, 3 cases reported by Nonne, 21 cases of hereditary ataxy reported by Sanger Brown, and one or two others.

More recently a monograph by Paul Londe has appeared, of which an excellent abstract has been made by Ormerod (85).

As the name denotes, the disease tends to affect several members of one family.

The leading symptoms are of the cerebellar type; namely, difficulty of maintaining the equilibrium, and an unsteady, reeling gait. The resemblance between the essential symptoms and those caused by experimental or other lesions of the cerebellum serves, according to Londe, to indicate the cerebellum as the seat of the disease; but the cases which have been examined after death are as yet too few to establish this with any degree of certainty. In one of those reported by Nonne there was general atrophy of all the cerebro-spinal centres, but there was entire absence of the postero-lateral spinal degeneration characteristic of Friedreich's disease (p. 152). In a case reported by Menzel there was great atrophy of the cerebellum, and degeneration also of the posterior columns, posterior roots, posterior root ganglia, crossed pyramidal and direct cerebellar tracts.

The chief points of distinction between cerebellar heredo-ataxy and Friedreich's disease are, first, that the knee-jerks are increased instead of being absent; and, secondly, that there are no trophic disturbances, and no scoliosis or talipes so characteristic of the latter disease. Londe is of opinion that this and Friedreich's disease both consist in a family tendency to degeneration of the cerebello-spinal system—the degeneration in the one group affecting chiefly the cerebellum, and in the other chiefly the cord, and sometimes, as in Menzel's case, both together.

Summary.—Lesions of the cerebellum may be entirely latent. This is the case most frequently when only one cerebellar hemisphere is affected.

The pathognomonic indication of cerebellar disease is an uncertain, reeling gait like that of alcoholic intoxication.

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LESIONS OF THE CEREBELLAR PEDUNCLES.—Lesions limited to the cerebellar peduncles are exceedingly rare, and in the case of the inferior peduncles practically unknown. The most exact information which we possess respecting the effects of lesions of these structures is derived from experimental physiology. The best-known experiments are those of Magendie on the middle peduncles. Magendie describes, as the chief effect of the section of the middle peduncle in the lower animals, an irresistible tendency to roll towards the side of lesion, and a peculiar skew deviation of the optic axes so that the eye on the side of lesion looked downwards and inwards, and the other upwards and outwards.

Curschmann has described as the effect of section of the united anterior and posterior cerebellar peduncles in rabbits, first, irregular muscular contractions, without rotation or change of position; and, secondly, a forced position to the side of lesion (Seitenzwangslage), to which the animal always returned if displaced, and which it retained till death. He obtained the same results on cutting through the middle peduncle separately, and attributes the rotatory movements and squint, described by Magendie, to lesion of the pons and tuberculum acusticum, rather than to lesion of the peduncle itself. Turner and I have found that division of any one of the cerebellar peduncles in the monkey produces symptoms similar to those of destruction of the lateral lobe (see page 363), the chief difference being the greater tendency to roll towards the side of lesion. The most characteristic and frequent forced position was observed after section of the inferior peduncle, namely, curvation of the vertebral axis, with the concavity towards the side of lesion; adduction and flexion of the limbs on the side of lesion, and abduction and extension of the limbs of the opposite side. In the majority of cases also the chin was deflected towards the side opposite the lesion, and in the direction of the rotation round the vertebral axis which caused the animal to roll to the side of lesion. The tendency to rotation, however, is only a temporary phenomenon, and ultimately entirely subsides.

Superior peduncle.—Curschmann has recorded a case of softening, from capillary hæmorrhage, in the right superior peduncle. There were, however, indications also of basilar tuberculous meningitis, which somewhat complicated the case. The symptoms were a forced position of the head and body towards the right side, to which position the patient invariably returned when resistance to the movement was withdrawn. There was no motor paralysis, and no deviation of the optic axes.

Bannister has recorded a case of minute hæmorrhagic extravasation in the ventral aspect of the right superior cerebellar peduncle. The symptoms were sudden onset of vomiting, which was very persistent, and a reeling gait. The patient lay on the right side with her head turned strongly to this side. The left arm was rigidly flexed, the left leg extended and lax. The eyeballs were in rapid horizontal movement from side to side. The whole of the left side became ultimately paralysed, and the right side also paretic. The patient in the later stages lay on her back, but the head was still turned to the right. Death occurred twenty-two hours after the onset.

Middle peduncle.—Lesions implicating the middle peduncle are not unknown, but they are rarely limited to the peduncle itself. Occasionally hæmorrhagic extravasation occurs, more or less limited to the peduncle, or extending also into the side of the pons.

The symptoms are somewhat variable, and it is not easy to eliminate the facts due to lesion of the peduncle from those due to lesion of the pons or adjacent structures. Not infrequently paralytic symptoms of the limbs and cranial nerves complicate those strictly dependent on the peduncular lesion. The most characteristic symptoms that have been observed are forced movements, or actual rotation, associated occasionally with deviation of the optic axes. A case, observed by Serres, has been reported by Longet, in which there was a focus of softening at the point where the right cerebellar peduncle radiates into the cerebellar hemisphere. The symptoms began with rotation from right to left, followed by left hemiplegia, which ultimately disappeared, with the exception of the paralysis of the left leg. Death occurred five months after the first onset. In another case, reported by Nonat, the patient, a woman aged 60, had an apoplectic attack due to hæmorrhagic effusion in the right middle peduncle. The patient assumed a forced position on the right side, with the head strongly turned to this side. The eyes were immovable, and the optic axes deviated so that the right eye looked downwards and outwards, and the left upwards and inwards. Death occurred on the following day. Another interesting case has been reported by Friedeberg of hæmorrhagic effusion into the left cerebellar hemisphere followed by meningitis due to traumatic lesion. In this case the patient suffered from vertigo, with a tendency in walking to go to the left, followed by repeated attacks of rotation round the vertical axis from left to right. The eyes were in a constant state of oscillation. On cessation of the rotatory attacks vertigo continued, and a sensation as if he were falling to the right. The whole duration of the symptoms was about five weeks. A case of tumour implicating the right middle peduncle has been recorded by Bernhardt. In this case the patient when lying in bed had a tendency to turn from right to left. The eyes looked upwards and to the left, and there was some degree of nystagmus. In standing and walking there was a tendency to fall to the left.

Inferior peduncle.—There are no clinical cases on record of lesions strictly limited to the inferior peduncle. I have already alluded to the characteristic forced movements and tendency to rotation which invariably occurred after section of the inferior peduncle in monkeys. Very commonly also anæsthesia of the cornea and face occurred on the side of lesion, owing to almost unavoidable division of the subjacent ascending root of the trigeminus. On experimental grounds I should be led to suppose that forced movements or tendency to rotation associated with anæsthesia of the cornea would be pathognomonic of lesion of the corresponding inferior peduncle of the cerebellum. I have not been able to find any record of such a combination of symptoms in man. A case has been recorded by Couty of a tumour, the size of a nut, affecting the left inferior peduncle; this, however, was complicated by tuberculous meningitis of the convexity and base. During life there were motor disorders on the side of lesion—at first pseudataxic, and later hemiplegic, the face being unaffected. There was no loss of sensation. Brissaud has lately recorded a case of tumour of the left restiform body which affected also the anterior segment of the left cerebellar hemisphere. The situation of the tumour was diagnosed during life. The symptoms were slowly increasing deafness in the left ear, convulsive tic on the left side of the face, stiffness and pain in the neck, retraction of the head, severe occipital headache and cerebellar gait. No forced movements or rotatory disturbances were observed in these two cases, nor does there appear to have been anæsthesia of the cornea or face on the side of lesion. Mendel, in reporting a case of Graves' disease in which there was atrophy of the left restiform body, attempts to establish a relation between this disease and lesion of the restiform body, and quotes the experiments of Filehne and others, from which it appears that symptoms resembling Graves' disease were induced in dogs and rabbits by section of the corpus restiforme. Turner and I, however, observed no such phenomena in monkeys; and in man integrity of the restiform body has often been established in cases of Graves' disease (*vide* Joffroy and Achard, Hezel, and Maude).

It thus appears that the most characteristic symptoms of lesion of any of the cerebellar peduncles are—rotation round the vertebral axis, or forcible impulse causing the patient to assume some fixed position. In lesions of the superior peduncle the cases of Curschmann and Bannister show a forced position on the side of lesion; in those of the middle peduncle, the cases of Serres, Friedeberg, and Bernhardt show rotation from the side of lesion; and that of Nonat forced position on the side of lesion,—facts which are all in harmony with the experiments of Magendie, as well as with those of Turner and myself. For the rotation from the side of lesion is essentially the same phenomenon as forced position, or rolling towards the side of lesion.

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LESIONS OF THE MEDULLA OBLONGATA.—The pathology of the medulla oblongata is unusually complex. Not merely is it primarily liable to the same lesions as affect other nerve-centres, but, more frequently, it is implicated in diseases primarily originating in the pons or cerebellum; it is also liable to be affected indirectly by intracranial disease in general, particularly by those forms which cause an increase of the intracranial pressure. Being also a kind of isthmus between the brain and spinal cord, it is subject to ascending or descending degenerations in consequence of cerebral or spinal lesions; and, being the centre of many complicated synergic muscular acts, its nuclei are subject to special forms of degeneration similar to those which affect the oculo-motor nuclei and anterior cornua of the spinal cord. With the indirect affections of the medulla oblongata in connection with various forms of intracranial disease, ascending and descending degenerations, and the clinical history of bulbar or labio-glosso-laryngeal paralysis, it is not the purpose of this article to deal, as these subjects are discussed under their respective heads. Attention will be mainly directed to the forms of disease primarily affecting the medulla oblongata, and to the symptoms on which the regional diagnosis can be based.

Hæmorrhage.—Hæmorrhage limited to the medulla is comparatively rare; more often the pons and medulla are affected together. It may be the result of disease, or due to injury, such as a blow on the head or back of the neck. Duret (3) has shown that in animals blows on the head, especially on the forehead and vertex, may cause punctiform extravasations in the medulla oblongata and base of the brain. Schulz has recorded a case of bulbar paralysis immediately following a blow on the back of the neck, presumably due to traumatic hæmorrhage. Not infrequently effusions of blood take place in the fourth ventricle, either arising from the pons medulla or cerebellum, or from inundations of the lateral ventricles gaining access by the aqueduct of Sylvius.

Hæmorrhage into the medulla, or into the fourth ventricle, is usually fatal. Death may be instantaneous, or within a few hours, in profound coma with stertorous respiration and occasional convulsions. Very rarely bulbar hæmorrhage gives rise to chronic stationary lesions with bulbar symptoms, but these are mostly due to thrombosis or embolism, or acute myelitis.

Softening.—Softening of the medulla oblongata, due to thrombosis or embolism of the vertebral arteries and their branches, is of common occurrence, and is the cause of what is termed apoplectiform bulbar paralysis. The arteries of the medulla oblongata, according to the researches of Duret (3), arise from the vertebrals, or from their branches the anterior spinal and posterior inferior cerebellar. One set of these vessels, the medial, enter the medulla in the middle line, and supply the nerve nuclei. The medial branches to the eleventh and twelfth nuclei arise from the anterior spinal. Those of the tenth, ninth, and eighth come from the basilar or upper end of the vertebral artery. The other set—namely, the lateral or radicular branches—enter the medulla with the nerve-roots, and supply also the nerve nuclei. The radicular branches to the bulbar nerves arise from the vertebral, except that to the spinal accessory which comes from the inferior cerebellar; and that to the hypoglossal which arises from the anterior spinal artery. Those to the facial or auditory nerves may come from the vertebral, or basilar, or both.

Embolic or thrombotic occlusion of these arterioles gives rise to softening of the centres to which they are distributed, and therefore causes acute bulbar symptoms. The extent and severity of the symptoms vary as the position and extent of the softening. Generally the onset is sudden as in hæmorrhage, but the course is more slow and the duration more chronic. As a rule there is no loss of consciousness, and this is sometimes considered as a point of distinction between hæmorrhage and softening, but it is doubtful whether it is trustworthy.

The symptoms caused by softening so induced will be considered presently.

Tumours.—Tumours may originate primarily in the medulla oblongata; but more commonly they invade the medulla from the cerebellum, pons, or base of the skull. Besides the special localising symptoms there are the general symptoms of intracranial tumour. Of these vomiting is usually specially well marked, while optic neuritis is not infrequently absent.

Cases have been recorded of tumour actually in the substance of the medulla without any of the characteristic symptoms in life (Wilks).

Abscess.—Abscess of the medulla is very rare. Eisenlohr has recorded two cases of abscess of the medulla—the one secondary to empyema, the other to cerebro-spinal meningitis. Chassel has recorded one secondary to acute infectious osteomyelitis.

Acute bulbar myelitis.—Under this head an affection is described of the nature of bulbar or labio-glosso-laryngeal paralysis, but of rapid onset, and probably due to acute inflammation of the bulbar nuclei. The symptoms set in generally with dysphagia, followed by paresis or paralysis of the soft palate, larynx, and tongue. The extremities are occasionally paretic, and paralysis of the face and ocular muscles is not uncommon. The temperature is occasionally elevated, the pulse always rapid, and death occurs in a few days or weeks, invariably preceded by paralysis of respiration.

Microscopical examination, in the few cases so examined, has revealed signs of inflammatory change in the bulbar nuclei,—petechial hæmorrhages, thrombosis, and small-celled infiltration.

Compression of the medulla.—Sudden compression of the medulla is not uncommon from fracture or dislocation of the atlas and axis, as in falls, hanging, twisting the neck, or diseased vertebræ. Usually death is instantaneous from respiratory paralysis—the heart often continuing to beat for an appreciable interval after cessation of all respiratory movements.

Gradual compression of the medulla oblongata may be caused by chronic disease and tumours at the base of the skull, aneurysm of the basilar or vertebral arteries, etc.

Oppenheim and Siemerling attribute compression symptoms in some cases to dilated atheromatous vessels. They are of opinion that arterio-sclerosis often leads to dilatation of the vessels without any apparent anatomical lesion, and yet the pons, medulla, and the issuing nerves may suffer in function from the pressure so exerted.

Symptomatology.—The pathognomonic symptoms of bulbar lesion are a conjoint affection of the extremities, and of one or more of the bulbar cranial nerves, with impairment of speech and deglutition, together with cardio-respiratory disturbances. In some cases of bulbar lesion the limbs only are affected, in which case no certain diagnosis can be made of the seat of the lesion. All four limbs may be paralysed; or, in other cases, the affection of the limbs may be of the hemiplegic order. With the limbs the cranial nerve most frequently implicated is the hypoglossal, more rarely the seventh or sixth. When the hypoglossal is affected the symptoms are impaired mobility of the tongue, with a greater or less degree of dysarthria. This combination of symptoms, however, may also arise from disease of the pons, and there are no features which clearly distinguish between the one and the other. The tongue and the speech are rarely, if ever, so affected as in progressive bulbar paralysis, nor has atrophy of the tongue, with diminished electrical contractility, been observed. An exemplary case of bulbar lesion with paralysis of the limbs on the one side, and of the tongue on the other, has been recorded by Madame Goukovsky. The autopsy revealed a focus of softening limited to the left olive and left pyramid, and destroying the greater part of the roots of the left hypoglossal. She refers to a similar case reported by Sougues, which was complicated, however, with paralysis of the sixth and seventh nerves.

In some cases the affection of the limbs is of a diagonal character, that is, the arm is affected on the one side and the leg on the other. This can only occur when the motor fibres for one limb are cut before decussation, and those for the other after they have crossed; in such a case the lesion must be situated at the decussation of the pyramids. An instance of this kind, due to a minute hæmorrhage in the lower part of the medulla oblongata on one side, has been recorded by Tildesley.

Turner (15) and I have shown that destruction of the tubercle of Rolando causes anæsthesia in the region of distribution of the fifth nerve on the side of lesion, and analgesia of the trunk and limbs of the opposite side. A case illustrating this combination of symptoms has been recorded by Senator; it was due to thrombotic softening in the outer postero-inferior aspect of the left side of the medulla oblongata. The symptoms were anæsthesia of the left fifth, and almost complete analgesia of the right side of the body.

In some cases of bulbar lesion an ataxic condition of the limbs has been observed. Goldscheider has recorded a case of lesion affecting the pyramids, upper part of the olives, inter-olivary layer, and nuclear region on both sides, but specially the left, in which the left arm was choreic, but apparently normal as regards sensation; while there was loss of the sense of position in the right arm. He is of opinion that this case shows that the tracts of the muscle-sense occupy a different region from those of other forms of sensibility—the former lying mesial to the hypoglossal nerves, the latter occupying the outer portion of the formatio reticularis; and Starr considers that the absence of ataxy in anæsthetic limbs is due to the fillet not being affected. These are points, however, on which further investigation is needed.

A symptom of great importance in the regional diagnosis of bulbar lesions is the occurrence of dysphagia or paralysis of deglutition. Dysphagia may also occur to some extent from lesion of the pons, especially when the tongue is affected; but complete paralysis of deglutition is a sign of direct or indirect lesion of the medulla oblongata. This may occur when the movements of the tongue are only relatively slightly impaired.

Paralysis of the soft palate on one or both sides is a frequent symptom, and with this there may be entire loss of the palate reflex. Together with the affection of the soft palate there may be hoarseness or aphonia from paralysis of one or both vocal cords.

The association of paralysis of the palate and of the vocal cords, and especially the absence of palate reflex, serve to distinguish true bulbar lesion from the pseudo-bulbar paralysis resulting from bilateral lesion of the articulatory centres of the cortex or lenticular nuclei.

The conjoint paralysis of the palate and vocal cords indicates lesion of the nucleus of the accessorio-vagus, or of its root fibres. In a patient recently under my own care there was a combination of paralysis of the right side of the face, right side of the palate, right vocal cord, right sterno-mastoid and trapezius, as well as the right glosso-pharyngeal. In this case there was probably lesion of the roots of the respective cranial nerves at their emergence from the medulla oblongata. The muscles of mastication have been affected in some cases. (See cases by Dixon Mann and Tildesley.)

Trismus has been mentioned by Joffroy as a characteristic symptom in apoplectiform bulbar paralysis, but, as shown by Nothnagel, it is not a common symptom.

Cardio-respiratory disturbances, such as irregularity of the heart, acceleration or retardation of the pulse, sighing, irregular or laboured respiration, intense dyspnœa often amounting to orthopnœa, coughing, hiccough or vomiting have also been observed not infrequently.

In a case reported by Glynn, of a small glioma in the middle line of the medulla at the level of the calamus scriptorius, there were present intense dyspnœa, paralysis of the diaphragm, vomiting, impaired movement of the lips and tongue, dysphagia, abolition of the palate reflex, and weakness in all four extremities, especially the arms. In this case there was also a trace of sugar in the urine.

Albuminuria and glycosuria have also been observed in connection with bulbar lesions, the latter more particularly with lesions of the floor of the fourth ventricle; but their association with acute bulbar paralysis is by no means constant.

De Jonge records a case of a small tubercle reaching from just below the left olivary body to the first cervical nerve, in which there was glycosuria, which he attributes to the lesion in question. He cites 11 cases of glycosuria with disease of the medulla, in all of which the lesion was considerably higher than in this case; and he concludes, therefore, that the diabetic centre itself was not involved, but only the fibres proceeding from it to the periphery.

Summary.—The characteristic symptoms of lesion of the medulla oblongata are impairment or paralysis of articulation, phonation and deglutition, with disturbances of the heart and respiration, and not infrequently paresis or paralysis of the limbs on one or both sides.

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D. F.

APHASIA AND OTHER SPEECH DEFECTS

Introduction.—The word "aphasia" is now commonly used as a general term including the widest variety of speech defects, and of defects which are dependent upon lesions in very varied parts of the brain. This I consider to be very undesirable, because the influence of Broca's discovery is still so strong that the majority of persons connect this term "aphasia" with a lesion of one definite part of the brain—the third left frontal convolution. It seems to me desirable, therefore, that the word "aphasia" should be limited as much as possible to the defect caused by damage to this region; and that different names should be applied to the speech defects due to lesions in other regions of the brain. Such a course will be adopted in this article.

It is now well understood that one hemisphere of the brain exercises a dominant influence in connection with language both spoken and written, and that for right-handed persons it is the left hemisphere which is thus potent. In left-handed persons, however, it has been commonly found that the right is the dominating hemisphere for speech purposes. Thus it would seem that whether the left or the right hemisphere is to take the lead in speech functions in any given person, depends mainly upon whether such person is right or is left-handed.

The degree to which in either case the second hemisphere intervenes is a matter of considerable importance, not only physiologically, but also from the point of view of the facility with which compensation and recovery may be brought about in many cases in which speech is lost or disturbed.

Forms of word memory.—The problems concerning speech defects are especially complicated because of the existence of four different kinds of word memory, each having its seat of registration in a definite part of the cerebral cortex. Thus there is (i.) an *auditory word-centre*, where the sounds of words are registered; (ii.) a *visual word-centre*, where the visual images of letters and words are registered; (iii.) a *glosso-kinæsthetic centre*, where the combined impressions which pass to the brain as a result of the movements of the lips, tongue, palate, larynx, and other parts concerned with articulate speech are registered; and (iv.) a *cheiro-kinæsthetic centre*, where the sensory impressions resulting from the movements concerned in writing are stored up. From the latter two centres outgoing fibres emerge, and descend as part of the pyramidal tract; those from the glosso-kinæsthetic centre going to the motor speech centres in the bulb, and those from the cheiro-kinæsthetic centre going to the motor centres in the spinal cord by means of which the act of writing is performed.

The four word-centres are only specialised portions of the general auditory, the general visual, and the general kinæsthetic centres. The latter correspond with the so-called motor centres of the cortex. But in my opinion motor centres do not exist in the cortex—the functions formerly ascribed to such centres being really carried out by sensory centres of kinæsthetic type. The portion of these centres concerned with articulate speech is situated in the posterior part of the third left frontal convolution, and perhaps part of the foot of the ascending frontal (Broca's region); the portion concerned with writing has been supposed (after Exner) to be situated in the posterior part of the second frontal convolution. This, however, is doubtful; and it is even uncertain how far the writing centre has a locus apart from the region in which impressions resulting from more general movements of the hand and arm are registered. The auditory word-centre is situated in the posterior half or two-thirds of the upper temporal convolution, with perhaps the posterior part of the second temporal convolution. The visual word-centre seems to be localised in the angular gyrus and in part of the supramarginal gyrus. These, at all events, are the regions which clinico-pathological research in

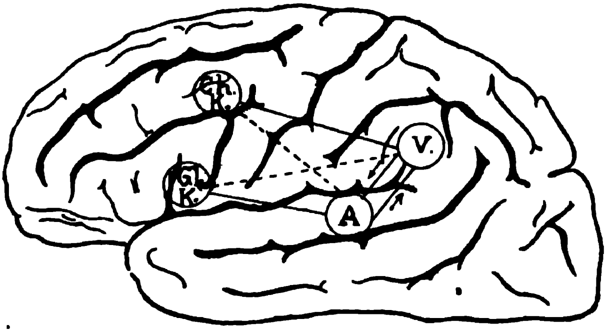

FIG. 22.—Diagram showing the approximate sites of the four word-centres and their commissures.

man seems to indicate as being most concerned with the several word-memories.

Order of development of the different word-centres.—In regard to the order of development of these different centres it would seem clear that the auditory word-centre is the first to be called into play, since the child learns to understand speech for many months before he is able to speak. After a time he makes attempts to speak, and then the glosso-kinæsthetic centre becomes gradually organised under the influence of incitations proceeding from the auditory word-centre—the stimuli passing along certain associational paths, which are thus gradually laid down. After a year or two the visual word-centre becomes organised as the child is taught to recognise letters and words; and simultaneously two sets of associational channels (or commissural fibres) are laid down between the auditory word-centre and this visual word-centre. Ultimately, when the child becomes able to read, there must be activity first in the visual word-centre, then in the auditory word-centre, and immediately afterwards in the glosso-kinæsthetic centre.

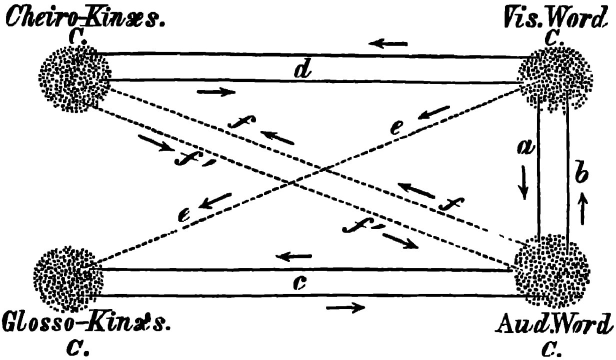

FIG. 23.—A diagram illustrating the relative positions of the different word-centres and the mode in which they are connected by commissures. The connections indicated by dotted lines indicate possible but less habitual routes for the passage of stimuli.

Subsequently, as the child learns to write, the cheiro-kinæsthetic centre becomes organised, the guiding influence of the visual centre being called into play, and this would be accompanied by the development of association channels between the two centres. The visual centre, in fact, holds the same sort of relation to the act of writing that the auditory word-centre holds to articulate speech. In writing from dictation, and probably also in writing spontaneously, the train of activity starts in the auditory word-centre, spreads to the visual centre, and thence on to the cheiro-kinæsthetic centre, whence the outgoing stimuli pass over to the motor centres in the spinal cord.

In these various ways functional associations of the closest kind become established between the four word-centres, those in the frontal convolutions (kinæsthetic centres), however, never acting alone but always n response to incitations coming to them either from the auditory or rom the visual word-centre.

The order of association above given is, however, probably not always observed. Thus there is reason to believe that in some persons who are “auditives” and well accustomed to write, the stimulus for this process may pass at once from the auditory word-centre to the cheiro-kinæsthetic centre without passing through the visual word-centre. And, again, that in certain well-educated “visuals” much accustomed to read aloud, the stimulus, during this process, may pass from the visual word-centre direct to the glosso-kinæsthetic centre without passing through the auditory word-centre—just as it must do in the case of deaf-mutes who have been taught to speak by the lip-reading process. These and other occasional associational channels are indicated in Fig. 23 by dotted lines.

The part played by the right cerebral hemisphere in speech functions.—Too little attention has been paid to this question, which becomes one of great importance when we have to consider the mode in which a cure, or restoration of function, is brought about in many varieties of speech defect. As above indicated, functional compensation in the case of destruction of the left auditory word-centre may sometimes be effected by the visual word-centre taking on new functions (that is, acting directly on the glosso-kinæsthetic centre); and in the case of destruction of the visual word-centre functional compensation may be brought about by the auditory word-centre taking on new functions (that is, acting directly on the cheiro-kinæsthetic centre).

But other possibilities in the way of functional substitution are to be looked for by reason of the word-centres in the right hemisphere assuming an unwonted degree of activity.

Some writers (Lichtheim, Wernicke, Déjerine) seem to think that no word-centres exist in the right hemisphere of a right-handed person. This is, in my opinion, a great mistake.

When we hear another person speak, the auditory impressions must impinge upon the right as well as upon the left hemisphere; and, similarly, when we read letters or words in a book, we know that such visual impressions must proceed equally to the two hemispheres. Again, when we speak, glosso-kinæsthetic impressions should stream up equally from the bilateral muscles in action to the third frontal convolution in each hemisphere. In writing with the right hand, cheiro-kinæsthetic impressions, of course, go only to the left hemisphere, so that here the case is exceptional, and the corresponding centre of the right hemisphere must remain undeveloped. The case of this latter function is therefore different from that of the other three whose performance seems necessarily to lead to development of the corresponding centres on each side of the brain. Thus it would appear that a certain amount of organisation of the right auditory word-centre, of the right visual word-centre, and of the right glosso-kinæsthetic centre, is certain to occur simultaneously with the organisation of corresponding centres in the left hemisphere. We seem bound to admit, therefore, that each hemisphere has the chance of being equally educated so far as the mere *reception of speech impressions* is concerned. It is when we come to the executive side of speech functions that the great difference begins to appear between the relative activity of the two hemispheres of the brain.

The fact that after a time the left hemisphere takes the lead, and that motor incitations pass off almost exclusively from it, probably leads gradually to a more complete organisation of the different word-centres in this hemisphere, and a more complete knitting of them together by the development of commissural fibres.

The similar centres in the two hemispheres are probably also brought into association with each other by means of commissural fibres forming part of the corpus callosum. The existence of such associational fibres between the two glosso-kinæsthetic centres accounts for the recovery of speech that takes place in certain cases of aphemia. The existence of similar fibres between the two auditory word-centres seems shown by the non-occurrence of word-deafness in cases where hearing is lost on the right side; whilst a consideration of the pathology of the condition known as "pure word-blindness" (as we shall see later on) makes it probable that commissural fibres also exist between the two visual word-centres.

The difference in the degree of activity of the two cerebral hemispheres during speech would tend to become more and more accentuated in future years, and would in all probability lead to a much higher grade of functional activity, even on the receptive side, in the three kinds of word-centres pertaining to the leading hemisphere.

It will subsequently be shown, moreover, that there is reason for believing that each auditory word-centre is in relation with Broca's centre, so that in some cases where there has been destruction of the left auditory word-centre word-deafness may be transitory, and fair speech soon be possible by reason of the increased action of the auditory word-centre of the right hemisphere, through a diagonally disposed commissure, upon the left glosso-kinæsthetic centre.

The seat of the primary recall of words.—In silent thought and in ordinary speech a memorial recall of words in the great majority of persons seems to take place first in the auditory word-centre, though this is almost immediately followed by less or more activity in Broca's centre. In a very small percentage of persons who are exceptionally strong "visuals," this primary recall of words may occur in the visual word-centre. All clinical evidence, however, is against the notion that the initial activity in the recall of words ever occurs in Broca's centre. This, like other kinæsthetic centres, seems only to be called into play at the instigation of stimuli coming from the auditory or the visual centres, though the contrary view has been strongly advocated by Stricker and others. This subject I have recently dealt with more at length in my first Lumleian lecture (5).

Different modes of incitation of auditory and visual word-centres.—It is important for the interpretation of speech defects to bear in mind that the functions of the auditory word-centre are carried on in obedience to stimuli of three degrees of intensity. (i.) The weakest stimuli, and the first to fail in producing their customary effect, are what we may call "volitional" stimuli, under the influence of which so-called spontaneous speech is produced—words being recollected as they are wanted for the expression of thought. (ii.) Stronger stimuli are those of an "associational" order, which come to the auditory from the visual word-centre when this is actively aroused, as in the process of reading aloud. (iii.) The strongest stimuli of all are those of a "sensory" type, coming to the auditory word-centre directly from without, as when a patient is bidden to repeat any given word.

Except in the very rare cases in which words are first revived in the visual word-centre rather than in the auditory during silent thought, the former centre reacts only to associational and sensory stimuli—so that what is known as *amnesia verbalis* (which is a failure of recollection for words) is practically limited to defects of the auditory word-centre.

Word-images as integral components of percepts and concepts.—The functional unification of the word-centres of which I have hitherto spoken is not of such a kind as to remain in a state of isolation. Words that are heard are first of all associated in the mind of the child with external objects, so that such auditory impressions become linked, by means of associational fibres, with the organic seats of the several sensory impressions—also freely connected with one another—that the child has been able to derive from this or that object. Thus the name subsequently becomes revived as part of the perceptive process whenever the object is again presented. The sight of the mother recalls the name "Mamma," just as the sound of this word would revive the corresponding visual, tactile, and other images. After a time the auditory representative of the name becomes reinforced by glosso-kinæsthetic impressions as soon as the child learns to utter the word; later by visual impressions when it learns to read, and by cheiro-kinæsthetic impressions when it has learned to write.

The several components of word-percepts are thus brought not only into relation with one another, but become no less intimately associated with the several sensory components of object-percepts. The result is that the hearing or the sight of the name of any object immediately calls up in the older child or in the adult an idea of the object, just as its presentation to sight or other sense produces a nascent activity in the several centres in which its name is registered, though mainly in the auditory and the visual centres.

Words soon become to a very large extent the symbols whereby we carry on our thoughts, and this thinking by means of words becomes all the more thorough as thought becomes more complex. We can think of a particular person or of a particular plant as well by recalling the visual image as by recalling the name. But when in thinking we have to recall general names, such as "animal" or "tree," or still more abstract names such as "virtue" or "vice," it becomes certain that in silent thought we use the words as symbols for the more or less complex ideas which they represent. And if we bear in mind that the seats in which these words are registered (our word-centres) are in organic and functional relation with the seats of registration of the corresponding percepts, concepts, etc., we are enabled in a measure to understand how the revival of the word in the mind (as the thought-counter) is associated with an almost simultaneous activity in the seats of registration of the corresponding percepts and concepts.

We may thus recognise how it happens that in simple acts of perception, and still more in simple thought processes, we have no limitation of cerebral activity to narrowly localised centres, but rather widespread processes of activity in very varied regions of the cortex, and that, too, in both hemispheres of the brain. In our talk about individual centres and their functions we are apt to forget how much the brain acts as a whole even in operations that seem comparatively simple, and many of those who have written on aphasia have, as I venture to think, not sufficiently taken into account the fact that the name constitutes an integral element of the percept or of the concept.

Does conception take place in a single centre altogether apart from perception?—This is a most important question concerning which much difference of opinion exists among writers on speech defects. Broadbent, Kussmaul, Charcot, Lichtheim, and Wernicke have taken the affirmative position more or less positively. I was the first to express dissent from this view, and have since been followed by de Watteville, Ross, Allen Starr, and Wyllie, all of whom have likewise decided against the supposed necessity of postulating a separate centre for ideas or concepts.

My opposition to this postulation of a single separate "centre for concepts" (or "centre for ideas" as it is called by others) was based originally upon psychological considerations. It seemed to me wholly unnecessary and at variance with what appeared to be the real nature of the process of perception and conception.

Then, again, I am unable to find any clear evidence from clinical data tending to prove the existence of a separate "centre for concepts"—or, in other words, any existing forms of speech defect that can only be explained by supposing the existence of a lesion in such a separate centre or, as Lichtheim supposes, in the course of its afferent or efferent fibres. I am convinced that the supposed necessity for assuming the existence of a single separate "centre for concepts," when seeking to interpret different forms of speech defect, may in many cases be obviated by a fuller recognition of the different degrees of functional excitability that may obtain in the auditory and the visual word-centres respectively. We shall see that their molecular mobility may be so much lowered that they are only capable of responding to powerful stimuli. Thus, whilst volitional recall or "recollection" may be impossible within their province, they may still be capable of acting in association with other centres—as when reading may be fluent though voluntary speech is greatly impaired; and they may act still more easily under a direct sensory stimulus

—as when a word is repeated which the patient has just heard pronounced.

Another cause that has led to this postulation of a "centre for concepts" is traceable, I think, to an inadequate realisation of the nature of a perceptive process, and of the fact that a name may and does constitute an integral part of the complexus of revived sensory impressions which go to constitute such a process.

Again, as it would be quite easy to show, perceptive processes vary greatly in complexity, and merge by insensible gradations into processes of conception. It seems thoroughly legitimate, therefore, to suppose that these latter more specialised modes of mental activity, whilst having their roots in perceptive centres, must be completed in outgrowths therefrom—that is, in parts of the brain which are in close relation structurally and functionally with the several sensory centres. I have commonly spoken of such regions as "annexes" of the perceptive centres.

Of late Flechsig (33) has called special attention to four areas of the cortex that differ from the sensory areas, since they are neither in relation with afferent nor with efferent fibres. He assumes that these regions subserve higher mental functions than those carried on in the sensory centres, and terms such regions "association areas." These regions seem, therefore, to correspond with what I have referred to above as "annexes of the perceptive centres." They occupy a large proportion of the cerebal cortex and are thus located by Flechsig: (i.) in parts of the pre-frontal lobes; (ii.) a large portion of the temporal lobes; (iii.) a considerable area in the posterior parietal region; and (iv.) the island of Reil. These four fairly well-defined areas are, as above stated, not directly connected with afferent or with efferent fibres, and in addition to this two other reasons are given for supposing them to be concerned with the carrying on of higher functions. Flechsig points out, in the first place, that these regions remain immature and completely devoid of myeline for several months after birth, though the sensorial centres have arrived at comparative maturity; and, secondly, that these association centres are the parts which are especially developed in the brain of man as compared with that of the lower animals.

It is only fair to Broadbent to point out that more than twenty years previously he had cited (18) almost exactly these regions of the cerebral cortex as parts that were neither in direct relation with peduncular fibres nor with those of the corpus callosum, and that he had attributed to these regions just the same functions as those now assigned to them by Flechsig. He adds: "Now the convolutions which I have enumerated as having no direct communication with the crus, central ganglia, or corpus callosum, are, in the first place, those which are latest in order of development, and on this ground alone might be supposed to be concerned in the more strictly mental faculties which are latest in their manifestations. They are those which constitute the difference between the human cerebrum and the cerebrum of the quadrumana; and it would, moreover, seem to accord with the general plan of construction of the nervous system and with what we know of the mental operations that these convolutions, which are withdrawn, so to speak, from direct relation with the outer world, should be the seat of the more purely intellectual operations."

It is, I think, perfectly legitimate to suppose that the annexes of the sensory centres, to which I have previously referred, tend to be developed in the directions above indicated by Broadbent and Flechsig, though how much of these territories they occupy must remain altogether uncertain. It seems also probable that there is no sharp line of demarcation between these annexes and the several sensory areas, and that *the combined sensory areas together with the annexes are accustomed to be thrown into functional activity more or less simultaneously.* Thus the processes of perception and conception, together with revival of linguistic symbols, are probably almost as inseparable in their localisation as they are in their nature and modes of occurrence, and their anatomical substrata must be supposed to occupy a very considerable extent of the cortex of both hemispheres.

A final question now remains for consideration related to this other which we have just been considering. It is this—Where are we to look for the registration and revival of words in the cerebral cortex? It may be said that this question has been already answered. And so it has tentatively and in a general sense. We have laid stress upon the existence of four different kinds of memorial registration of words and the probable sites of such word-centres in the hemispheres. We have indicated also that the glosso- and the cheiro-kinæsthetic centres constitute definite parts of the general kinæsthetic centres, and that the auditory and the visual word-centres probably also constitute more or less separate parts of the general auditory and visual centres.

Something additional, however, may now be said concerning the sites of these word-centres which could not well have been said at an earlier period, and that is, that each of them is probably to be found partly on the confines of its percept centre and partly on that of its related annexe. This supposition is made because some words (especially names of things, persons, and places) are in closest relation with sensory centres; whilst others, such as verbs, adjectives, prepositions, and other parts of speech constituting the framework of language, are in closer relation with conceptual processes. These two modes of functional activity are, as I have said, absolutely inseparable from one another, and therefore the several word-centres must be in most intimate relation both with the sensory centres and with their annexes.

If the views above expressed be anything like an approximation to the truth, it may be judged how vain it would be to attempt to base our explanation of any of the different kinds of speech defect upon the supposed existence of some one separate centre for "ideation," "conception," or "naming," which is connected by means of commissures (long enough and separate enough to permit of isolated damage) with sensory centres on the one side and with motor centres on the other.

The two writers who have, in recent years, dwelt most in their enumeration and classification of speech defects upon the supposed existence of a separate centre for concepts have been Lichtheim and Wernicke—the latter to a considerable extent following, though also developing, the views of the former. Lichtheim's memoir, published in 1884, and illustrated by diagrams in which he shows a centre for concepts altogether apart from the sensory centres, attracted great attention both in this country and abroad. He described forms of speech defect which he attributed to lesions of the afferent and of the efferent fibres respectively in relation with such a centre for concepts—and in this he was followed by Wernicke. Yet when we seek Lichtheim's justification for thus postulating a centre altogether apart, and embodying this view in numerous diagrams, we actually find him saying (46) "this has been done for simplicity's sake," and that he does not consider the function of conception "to be localised in one spot of the brain, but rather to result from the combined action of the whole sensory sphere." This statement seems to me to invalidate much of his exposition, and to make it almost impossible for him legitimately to suppose, as he does, that two of his types of speech defects are to be explained by the supposition of the existence of a lesion involving either the afferent or the efferent fibres pertaining to such a widely-diffused centre. In addition there is the serious defect that his diagrams are at variance with his views on this important subject.

Classification of Speech Defects

From what has been said it will be seen that in the study of speech defects it is necessary to consider the effects of lesions in the following situations: (*a*) in the different kinds of word-centres; (*b*) in the different commissures by means of which these centres are connected with one another; (*c*) in the internuncial or pyramidal fibres connecting the two kinæsthetic word-centres with their related motor-centres, in the bulb and in the cervical region of the spinal cord; and (*d*) in these motor-centres themselves which are concerned with the actual production of speech and writing.

On the whole, however, it seems best to describe the defects in a somewhat different order. We shall begin, therefore, with the consideration of defects due to lesions in the motor-centres; then to lesions of the pyramidal fibres; and, lastly, to cortical lesions involving the different centres and the commissures by which they are united. Our subject will consequently be dealt with under the following main divisions:—

(A) Sub-cortical lesions:—I. Lesions of motor-centres for speech and writing; II. Lesions of pyramidal fibres going to these centres. (B) Cortical lesions:—III. Lesions of the glosso- and cheiro-kinæsthetic centres; IV. Lesions of the auditory and the visual word-centres; V. Lesions of the commissures between the word-centres.

I. DEFECTS OF SPEECH AND WRITING DUE TO STRUCTURAL OR FUNCTIONAL DEGRADATION IN MOTOR-CENTRES

Loss or extreme difficulty in *speech* due to structural disease in the bulbar speech-centres is now commonly known by the name *anarthria.* In the different forms of this trouble we have to do with various grades of difficulty in utterance, while the power of communicating the thoughts by writing, as well as the mental power of the patient, may be unimpaired. It is, at all events, unnecessary that there should be any accompanying defects of this kind. Both spoken and written speech may also be comprehended as well as ever.

There are two general diseases of the nervous system, namely, chorea and disseminated sclerosis, in which slight anarthric defects are commonly met with. And there are other localised diseases of the bulb in which structural lesions of a better known and of a grosser type exist, and where difficulties of utterance are more uniformly encountered. These diseases include the chronic affection known as glosso-labio-laryngeal paralysis, and the various acute affections comprised under the term "acute bulbar disease."

It is quite unnecessary to dwell upon these different forms of difficult utterance or anarthric defect, because a reference to their several characters will be found under the description of these different diseases.

Other speech defects of the anarthric type are due to functional perversions rather than to structural defects of the motor speech-centres. Of these there are three principal varieties, namely, (*a*) Lalling; (*b*) Stammering; and (*c*) Aphthongia.

(*a*) *Lalling* is, as Kussmaul says (43), "the term used to characterise the speech of children before they have learned to pronounce their words so as to be intelligible to all persons." It is therefore a defect due to a want of precision in the oral articulatory mechanism. In an ordinarily healthy child who is properly taught, and who shows an adequate amount of attention, this want of precision soon disappears; but where the attention and training are defective it may linger long beyond the usual age. On the other hand, in those children whose brains and intelligence are defective this imperfect articulation may never wholly disappear. As a temporary defect, lalling is met with in persons under different degrees of alcoholic intoxication.

(*b*) *Stammering* is so important a defect that it is considered in a separate article [see page 448].

(*c*) *Aphthongia* is a very rare affection of which only a few instances have been recorded (Fleury, Panthel, Ball). According to Fleury (34), who first named and described the condition, its most prominent feature seems to consist of "cramps in the territory of distribution of the hypoglossi, which set in whenever an attempt to speak is made, and render articulate expression impossible." The condition has mostly shown itself after great mental excitement, and this on one occasion was due to an operation for excision of the tonsils. The spasm is sometimes limited to the intrinsic muscles of the tongue, though in Panthel's case the extrinsic muscles in the neck connected with the larynx were also involved. In Ball's case (1), when the patient attempted to speak there was a spasmodic contraction of the muscles of the tongue, causing the organ to assume a dome-like shape and to be closely applied to the roof of the palate.

A very few words will suffice for all that need be said concerning defects in *writing* due to structural or functional degradation of the motor-centres in the spinal cord upon which such acts depend.

It is obvious that structural disease involving the motor-centres in the spinal cord concerned with the act of writing will lead, through different degrees of paralysis of the hand and arm, to defects in this power comparable with different degrees of anarthria caused by lesions in the bulbar motor-centres. Disease of the peripheral nerves leading from the spinal centres would produce very similar disabilities in regard to writing—they are, in fact, sufficiently common in cases of peripheral neuritis—though well-marked defects of speech caused by disease of peripheral nerves is much less common.

Again, in writer's cramp we have a spasmodic and disordered action of the muscles concerned in writing, due probably to a functionally perverted activity of these spinal motor centres, leading to disabilities in connection with the act of writing which are very analogous to stammering and aphthongia as speech defects.

II. Defects of Speech and Writing due to Structural or Functional Degradation of Pyramidal Fibres

The fibres to which reference is now to be made are those in relation with speech that issue from the third frontal convolution and pass through the internal capsule to the bulbar speech-centres; while in the case of writing they are those that issue from the foot of the second frontal convolution (or thereabouts), and which similarly pass down in the pyramidal tract to certain motor-centres in the right side of the cervical enlargement of the spinal cord. To such fibres, connecting sensory with motor centres, I am accustomed to apply the term "internuncial," in order to distinguish them from "commissural" fibres, which connect either two sensory or two motor centres with one another.

The speech defects belonging to this category I have for some years past thought it best to group under the term *aphemia*. They vary very much in degree in different cases—that is, from mere "thickness" or indistinctness of articulation (incomplete aphemia) to actual loss of speech or dumbness (complete aphemia).

The cases of incomplete aphemia cannot, so far as the mere form of the speech defect is concerned, be strictly differentiated clinically from forms of anarthria of similar severity. The distinction between aphemia and anarthria must, therefore, depend upon the nature of the collateral associated conditions. One of the most important distinctions is the fact that the impaired utterance in bulbar disease is much more apt than in that due to damage to pyramidal fibres (except where this damage is situated in the pons Varolii) to be associated with more or less difficulty of deglutition, and also with more severe paralysis of the sixth, seventh, eighth, or twelfth cranial nerves. There is also, in the speech defects due to bulbar lesions, a much greater tendency to the existence of some amount of bilateral paralysis of limbs and trunk muscles.

It is, however, useful to keep up a difference in name founded upon the difference in the site of the lesion in the two cases. This course will contribute to clearness in the classification of speech defects.

In all the cases of aphemia, whether slight or severe, the essential characters of the speech defect are, as I have said, just like those of anarthria—that is, there may be no trace of mental impairment; where the right hand does not chance to be paralysed the patient's power of expressing his thoughts by writing is in no way diminished; and he is perfectly able to understand both spoken and written speech.

So far as we know at present, the internuncial fibres with which we are now concerned pass downwards on each side through the "internal capsule" in the situation of the *genu*, forming a slender band named by Brissaud the "geniculate fasciculus,"[1] which descends in the foot of the peduncle, between the internal and the middle third of this body, on its way to the nuclei in the bulb that are concerned with the movements of the lips, tongue, soft palate, and larynx in the production of articulate speech.

Similar amounts of damage to these fibres alone ought always to produce similar degrees of aphemia, in whatever part of their course the damage may occur. The varying degrees of aphemia met with in different cases will depend, therefore, upon the extent to which the functions of the fibres (all or some) are interfered with in some part of their course.

Aphemia is clearly not a sensory defect—it is not a form of amnesia—because the subjects of it can revive words in all possible modes, and are, therefore, able to think and express their thoughts with an unimpaired freedom by writing. If the aphemia be in any way incomplete, moreover, such a case can be easily discriminated from a case of aphasia by the fact that the aphemic patient will always at once make an attempt, when bidden, to pronounce some simple word or syllable (however poor the attempt may be), while the typical aphasic patient is unable to make any such attempt—he will not try even to repeat the simplest vowel sound.

The reason of this important distinction seems to me to lie entirely in the situation of the lesion in the two cases. In aphasia, one of the most important word-centres for the expression of thought is affected; whilst in aphemia, as I understand it, all the centres in which the memory of words can be revived are intact, the damage occurs beyond these, and there is consequently nothing to interfere with the flow of thought and, in incomplete cases, nothing whatever to prevent attempts at articulation being made, just as similar attempts can always be made by patients suffering from disease which involves the bulbar articulatory centres.

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[1] And by Pitres "le faisceau pédiculo-frontal inférieur"—which seems altogether too unwieldy a nomenclature.

Then, again, the degree of severity of the aphemia that would be occasioned by a given amount of damage to these fibres when it occurs in the right hemisphere of the brain is altogether less than that which would be produced by a similar lesion in the left hemisphere, so long as the person is right-handed. This is now fully recognised, and the reason for it was thus clearly stated by Kussmaul (44):—"The familiar fact that centro-hemispheric hemiplegia of the right side is often associated with defects of articulation, frequently amounting to total loss of speech, which are at once more lasting and more severe than the slighter and more transient dysarthries occurring with left hemiplegia, proves that the main current of the centrifugal impulses of speech passes downwards through the left cerebral hemisphere. But since dysarthric troubles—though usually of a trifling kind—are noticed, as a rule, in connection with left hemiplegia likewise, we must conclude that, beside the main current just referred to, a weaker accessory current is transmitted through the right hemisphere."

By the "weaker accessory current" of centrifugal impulses above referred to by Kussmaul as passing down through the right hemisphere, he meant the impulses that are transmitted from the right third frontal convolution through internuncial fibres on that side; and this view must not be confounded with that of Lichtheim, who assumes (in my opinion, on no adequate grounds) that the geniculate fasciculus from the foot of the third left frontal bifurcates rather high up, and sends a small contingent of its fibres by way of the corpus callosum to the opposite hemisphere, there to pass down in the right internal capsule (47*a*). He makes this supposition with the view of accounting for the more complete cases of aphemia (what he calls "subcortical aphasia") mostly found to be due to lesions situated in the white matter, not far below the cortex, in the region of the third frontal convolution.

It seems, however, quite unnecessary to make such an assumption, because these cases of complete aphemia due to subcortical lesions may be even better explained by supposing that we have to do with destruction, not only of the whole of the geniculate fasciculus, but also of the first part of the commissural fibres which pass from the left to the right third frontal convolution. It seems pretty clear that a lesion of these two sets of fibres would produce complete aphemia or dumbness. Two typical cases with lesions in this situation, in which there was complete mutism in one and almost complete mutism in another, whilst ability to write freely was preserved in both, have been recorded by Déjerine (25).

It would seem, however, that in some elderly persons an almost similarly complete aphemic condition may be produced by a lesion of the left geniculate fasciculus at some distance from the third frontal convolution—that is, close to the corpus striatum. Two such cases have been recorded by Pitres (58), and one by Oulmont. It looks as if in these cases, having regard to the sites of the lesions, the commissural fibres between the two third frontal convolutions could not have been involved, and that the complete aphemia must have been produced by the destruction of the left geniculate fasciculus alone.

Numbers of cases of lesions in the course of this left geniculate fasciculus will be found recorded as cases of "aphasia," or, in more modern language, as cases of "motor aphasia." Several of them are referred to by Pitres and Boyer, and others have been more recently cited by Ross, but very few of them afford any conclusive evidence. Some are doubtful cases, where there has been more than one lesion; others are cases in which the lesion was too extensive to be at all conclusive; whilst others, again, are cases in which the clinical details are too defective to enable us to speak positively as to the nature of the speech defect.

In cases of incomplete aphemia the patient's utterance (so far as it goes) is consistently and uniformly bad. In an aphasic patient, on the other hand, in whom the third left frontal is destroyed, there is often the constant distinct articulation of certain words or phrases ("recurring utterances"), the production of which is commonly attributed to the activity of the right hemisphere. Why then, it may be said, does the right hemisphere not similarly come into play in bad cases of aphemia? The only reply I can make to this is to suggest that when Broca's region is uninjured, and the speech stimulus is able to pass therefrom through a varying length of its own internuncial path without much obstruction, the right hemisphere is not called upon, and the speech stimulus continues to be sent along the old obstructed or blocked path. But when Broca's centre is destroyed, any speech that can be produced must result from calls upon the right hemisphere, and for long can only be such familiar and well-worn utterances as "yes" and "no," or common occasional expressions such as "nurse," "oh dear," etc.

In regard to the duration of aphemic defects this will depend upon two or three conditions. Much, for instance, will depend upon whether the fibres of the geniculate fasciculus are actually destroyed or only temporarily disabled. When they are only pressed upon by extravasated blood or an abscess, the subsequent partial absorption of the clot or the opening of the abscess may relieve the fibres from pressure, and so diminish the aphemia, while no such speedy disappearance of the speech trouble would be possible if these fibres were actually torn across. In these latter cases, however, there is still the possibility of the right hemisphere after a time taking on compensatory functions in the manner first suggested by Broadbent, that is, through incitations passing across by fibres of the corpus callosum from the left to the right third frontal convolution, and thence through the originally smaller right geniculate fasciculus to the motor-centres in the bulb. There are difficulties in the way of such a process, though it does seem to occur occasionally. A good example of this would appear to be furnished by a case published by

Déjerine (27), and also in another recorded by Pitres (59). In other cases, however, even where the damage to the left side of the brain has occurred in early life, so that the chance of the establishment of such a compensatory activity on the part of the right hemisphere might be regarded as greatest, no recovery of speech has taken place.

It is, of course, self-evident that if these internuncial fibres are damaged on both sides of the brain, there must necessarily be incomplete or complete aphemia, according to the degree of completeness of their destruction. In such a case, moreover, with severe bilateral lesions, recovery would be impossible. The clinical characteristics of this condition very closely resemble that produced by simultaneous lesions of the left together with the right third frontal convolutions, and they have been grouped together under the name of "pseudo-bulbar paralysis," because of their resemblance to the group of symptoms commonly met with where disease actually invades the bulb. A typical example of the double cortical lesion has been recorded by Barlow. In other cases a lesion of the centre has been met with on one side and of the internuncial fibres on the other.

All these cases are distinguished from those of real bulbar paralysis from the fact that there is a history of two separate attacks, and that it was after the second of these that the paralysis of the lips, tongue, and pharynx occurred. Then, in addition, in pseudo-bulbar paralysis there is no wasting of the tongue or alteration in its electrical excitability, and there is also no loss of the palatal or pharyngeal reflex. Still, in many cases, especially where the previous history is difficult to obtain, it may not be easy to arrive at a positive diagnosis between these two conditions.

No isolated defect of *writing* corresponding with aphemia as a speech defect seems to exist in a recognisable form. This is due to the fact that a lesion involving the internuncial fibres between the cheiro-kinæsthetic centres and the motor-centres in the cervical region of the cord, in any part of their extent, would almost certainly cause paralysis of the hand also for movements other than those concerned with writing. Thus the mere agraphic defect would be merged in and concealed by a wider form of paralysis. The reason why this same kind of result does not occur in the aphemic class of cases is because here, even though the internuncial channels may be blocked in the left hemisphere, by means of which the specially combined movements needed for articulate speech are called into play, the right hemisphere is still capable of calling into action the bilateral bulbar nuclei concerned with other less specialised movements of lips, tongue, and palate; consequently, it is only the articulatory movements of these organs that are paralysed in a case of aphemia. The special paralysis of speech is not merged in a wider defect simply because, for the actuation of movements other than those of speech, the bilateral motor-centres of the medulla may still be called into play by the undamaged right hemisphere.

III. DEFECTS OF SPEECH AND WRITING DUE TO DISABILITIES IN THE GLOSSO-KINÆSTHETIC AND CHEIRO-KINÆSTHETIC CENTRES

It will be best first of all to consider the effects of ordinary lesions in these centres. We shall thus be the better able subsequently to consider the effects of mere functional disabilities and transitory disturbances of these centres brought about by a great variety of causes, and which consequently are by no means uncommon.

Defects due to gross lesions.—These kinæsthetic centres are, as I have already observed, concerned more with the expression of thought than with the thinking process. Their activity is in the main roused as thought is about to translate itself into action. Although with lesions limited to these regions the power of thinking may not be very greatly interfered with, still it is nearly always interfered with to some extent, so that patients having such lesions do not usually exhibit anything like the same amount of mental clearness as that shown by patients suffering from aphemia.

The fact that speech and writing are so frequently involved together in cases of *aphasia* is due partly to the proximity of the glosso- and the cheiro-kinæsthetic centres, and perhaps not less to the proximity of the two sets of commissural fibres connecting these centres with the auditory and the visual word-centres respectively. Simultaneous damage to these commissures may in fact be a cause of typical cases of aphasia with agraphia, not distinguishable in their clinical characteristics from the results of combined disease of the centres themselves.

Some of the earlier observers of aphasic cases, such as Trousseau, Hughlings Jackson (39), and Gairdner, seemed to consider that the inability to write was as much a result of a lesion in Broca's region as inability to speak, and contended that there was almost always a parity between these defects. This, however, is not borne out by an examination of recorded cases, as I have endeavoured to show in my second Lumleian lecture (6).

One of the first to dwell upon the marked inequality that may be met with between these two disabilities was W. Ogle, to whom we owe the introduction of the term "agraphia." This latter kind of defect is much less noticeable than the former, because the patient is also often more or less paralysed in the right hand and arm. In such cases attempts at writing would only be possible, if at all, where the right side of the brain and the left hand have been more or less educated, and frequently no serious efforts have been made in this direction.

Though aphasic patients are unable to give voluntary and preconsidered expression to their thoughts, words, or even short phrases and oaths, may occasionally be uttered under the influence of strong emotion. We often find these patients able to make use of short familiar words like "yes" or "no" in response to questions addressed to them, though they may be often inappropriately employed. The articulation of such words, or "recurring utterances" as they are now commonly termed, is generally supposed to be brought about through the intervention of the comparatively uneducated right third frontal convolution. As Hughlings Jackson originally pointed out, such a patient is quite unable to repeat one of these words which he is continually bringing out, or, indeed, any other simple vowel sound when he is asked to do so. He cannot utter it, that is, in a purely voluntary manner, in response to a request or command. Another interesting peculiarity is also often seen when resident foreigners become aphasic. During recovery it is found that they are at first only able to express themselves in that language in which they are most thoroughly versed—namely, in their own native tongue. I have seen this in several patients. Two were Germans who had been long resident in this country; yet, after an attack of right hemiplegia and aphasia, each of them was for a long time unable to utter a word of English. When they began to speak they used German words only; and after they had further recovered, if occasionally in want of a word while speaking English, it was always a German equivalent that first presented itself.

There are many instances on record in which, though the aphasic condition itself has been complete and associated with more or less agraphia, the mental powers of the patients have been fairly well preserved. Many of such individuals have been able to read intelligently to themselves, and play games, like draughts or cribbage, perhaps better than their neighbours.

The views of Trousseau and others before referred to, who maintained that agraphia as well as aphasia is a consequence of destruction of Broca's region, have of late years been adopted by Wernicke, Déjerine, Wyllie, and Mirallié. They go farther still in attaching an overweening importance to this centre. They maintain that destruction of this region causes, in addition to agraphia, an interference with silent thought by hindering the revival of auditory word images, and that it also leads to alexia. Wernicke, Déjerine, and Mirallié even disbelieve in the existence of any cortical centre having the same relation to writing movements that Broca's centre has to speech movements. These various points require some separate consideration.

(i.) *Does destruction of Broca's region alone entail verbal amnesia?*—To this question I am decidedly inclined to give a negative answer, relying upon the reasons previously given to show that words are primarily revived in the auditory word-centre rather than in Broca's centre, and upon the evidence of actual cases to show that where the lesion is limited to this region there is no sympathetic disturbance of the auditory centre sufficient to cause amnesia verbalis after the first shock that may arise from the lesion has subsided. Cases recorded by Guido Banti, Dickinson, Wadham, and others (see second Lumleian lecture) fully bear out this view, seeing that the ability of such patients to write freely showed clearly that they could recall words. Exceptions to this rule may occasionally be met with; that is, it may happen that the severance of Broca's centre from the auditory and the visual word-centres may greatly impair the functional readiness of these latter centres, and so hinder the spontaneous revival of the auditory and the visual images of words. This, however, I believe to be the exception rather than the rule.

(ii.) *Does destruction of Broca's region entail alexia?*—In reading, a proper comprehension of the meaning of the text requires, as I believe, a conjoint revival of the words in the visual and in the auditory word-centres, but that for this mere comprehension it is not necessary for the stimulus to pass on to Broca's centre as it must do in reading aloud.

It may, however, be freely admitted that if the way is open, and this latter centre is in a healthy condition, it does commonly receive in reading to oneself a slight stimulus from the auditory word-centre, a fact which is often enough shown by the occurrence of involuntary, half-whispered mutterings when reading. It may also be admitted that the rousing of all three centres does give assistance in the comprehension of anything difficult, as is shown by the common practice of reading aloud any passage the meaning of which may be at all obscure. By this proceeding aid is obtained, however, not alone by the full rousing of the glosso-kinæsthetic centre, as there is also the full activity of the auditory word-centre following upon the spoken words. This kind of aid is indeed commonly needed, as Wernicke pointed out, by an uneducated person for the full comprehension of almost all that he reads. Therefore it may easily happen when such a person becomes aphasic from destruction of Broca's region that alexia may go with the aphasia. Similar aid may be necessary in cases in which there is functional weakness of the visual word-centre. It would seem, however, that in some persons the comprehension of writing or printing does not even require the associated activity of the auditory word-centre, so long as the visual word-centre is in a healthy condition and its other associational fibres are intact. This is proved by the fact that in a case recorded by Wernicke and Friedländer, in which there was aphasia associated with word-deafness (due to a lesion in Broca's region and also in the upper temporal convolution), the patient was able to read well and even to write. There is also a valuable case of word-deafness (with lesions in both upper temporal convolutions) recorded by Mills, and to be referred to farther on, in which the patient was able to understand what she read; and a still more remarkable case recorded by Pick, with very similar lesions, in which, although the patient was word-deaf, he could write and comprehend perfectly what he read.

Many of the cases in which there has been alexia coexisting with aphasia are, I think, instances where there have been rather wide lesions, extending far beyond the third frontal convolution and involving also the visual word-centre. Some of them may be cases in which there was originally word-deafness as well as word-blindness, but where the former has disappeared early—which Lichtheim contends to be customary. I have shown, moreover, that in some of the cases in which alexia coexists with aphasia, hemianæsthesia is also present. This fact would tend to prove that in such cases we have either to do with a wide lesion extending from the third frontal convolution back to the posterior part of the internal capsule, or else with a separate lesion in this situation—that is, in a brain region contiguous to the visual word-centre, which might therefore itself have been involved.

(iii.) *Is it right to deny the existence of a special centre for the registration and regulation of writing movements?*—The case in regard to writing seems to me to be almost exactly comparable with that of speaking, and indeed of all other habitual voluntary movements, whether complex or simple. Each set of movements must be associated with a set of ingoing impressions (kinæsthetic) which are registered in different portions of the Rolandic area of the cortex. This must be as inevitably true for writing movements as for speech movements; and just as re-excitation of Broca's region under stimulation from the auditory word-centre is necessary for speech, so a re-excitation of the centre in which the impressions generated by writing movements are registered is, under stimulation from the visual word-centre, needful for the production of writing movements.

Yet both Wernicke and Déjerine say that the intervention of a special centre for writing movements is not necessary, on the ground that writing consists of a simple copying of the visual images stored up in the visual word-centres. But I maintain that the visual word-centre cannot act directly upon the motor-centres in the cord, and that the conjoint activity (for co-ordinating purposes) of a kinæsthetic centre is quite as essential in the case of writing movements, as is the activity of Broca's region for the production of speech movements.

Again, how can it possibly be alleged as a valid argument against the existence of a cheiro-kinæsthetic centre in a man accustomed to write with his right hand that this same man is able to write rudely on sand with his foot, or that he can, after a fashion, write with his elbow, or with a pencil placed between his teeth? Clearly, in these other crude modes of writing he would simply be dependent upon the activity of other parts of the general kinæsthetic centre. Such objections seem, therefore, to be altogether futile.

It is, however, altogether another question, and one which does seem open to discussion, whether the cortical centre in which the sensory impressions produced by writing movements are registered exists altogether apart, or whether its structural elements are inextricably mixed up with others pertaining to less special movements of the hand and arm. If there is a completely separate seat of registration, destruction of such a centre should produce loss of the power of writing, independently of paralysis of other less special movements of the limb. If otherwise, the loss of power of writing from damage to the cortical region in which its kinæsthetic impressions are registered would never be able to occur alone, but would always be merged in a more general paralysis of the limb, and it will be remembered that we came to the conclusion that this was the cause of the absence of agraphia as a separate symptom resulting from damage to the pyramidal tract. In other words, no agraphia of this type would be recognisable as such; it would not be looked for, because the coexisting paralysis of the hand and arm would naturally be supposed to be, and would in fact be, a sufficient cause of the inability to write. On the whole it must, I think, be said that no actual proof has yet been given that the centre for writing movements is topographically distinct—in other words, that Exner's or any other separate localisation of such a centre has yet to be definitely proved. The evidence that I have previously referred to, though it has been sufficient to discountenance the idea that destruction of Broca's centre of itself gives rise to agraphia, has no pretence to be adequate for the localisation of a separate centre, and the cases that have hitherto been published in support of Exner's localisation are all of them open to very legitimate objections, as I have endeavoured to show elsewhere.

Then, again, one of the causes which led Déjerine to deny the very existence of a centre for writing movements seems to have been based upon the characteristics of two cases that were brought forward by Pitres, and which, upon the basis of his authority, have been often cited by others as affording some evidence of the correctness of Exner's localisation. They were cases in which the patients were unable to write spontaneously or from dictation, and yet were able to copy writing. If the centre is destroyed, says Déjerine, the possibility of executing any kind of writing should be abolished. That at first sight seems perfectly true; and it may be a valid criticism against Pitres for quoting any such cases as instances of "motor agraphia." But Déjerine and his followers have overlooked the possibility that Pitres may not have been quite correct in his interpretation of these cases—in which there was no necropsy. They have not recognised the fact that this particular combination of symptoms may be present without the existence of what is called "sensory aphasia" in any of its forms. It can be shown, however, that such a combination of symptoms may be easily explained by a cutting across of the audito-visual commissure that transmits impressions from the auditory to the visual word-centre (Fig. 23, *b*). This I believe, after careful study of his memoir, to be the explanation of the partial agraphic defects in the two well-known cases cited by Pitres, as examples of the effects produced by damage to the cortical centre for writing movements (60). It is obvious that damage to the commissure above mentioned would be capable of preventing the passage of stimuli from the auditory to the visual word-centre such as would be necessary in writing spontaneously or from dictation, whilst it would leave the power of copying writing intact so long as the visual word-centre, the commissure connecting it with the writing centre, and this centre itself remained undamaged.

The question, therefore, whether there is an altogether separate cheiro-kinæsthetic centre must be considered as still doubtful, and as there are no simple uncomplicated cases of agraphia, instances of this defect in association with aphasia to some minor extent can alone be referred to in illustration of damage to a possibly separate cheiro-kinæsthetic centre, or from damage to the commissure connecting it with the visual word-centre. This is the class of cases that was originally reported by W. Ogle in his paper on "Aphasia and Agraphia." In these cases there is no association with word-blindness, and nothing to prevent the patient learning to write with his left hand. Two such cases have been recorded by Broadbent (19), and one by Ogle.

The way in which these forms of agraphia are to be distinguished from the agraphia occurring under other conditions may best be shown by the following table, in which are embodied conclusions the legitimacy of several of which will be subsequently shown :—

AGRAPHIA

Complete	Uncomplicated.	1. Destruction of cheiro-kinæsthetic centre. 2. Destruction of the visuo-kinæsthetic commissure.
	Complicated with word-blindness.	3. Destruction of the visual word-centre.
Partial	No spontaneous writing or from dictation, but can copy; no word-deafness.	4. Destruction of the audito-visual commissure.
	No spontaneous writing, or from dictation, but can copy; word-deafness present.	5. Destruction of the auditory word-centre.
	Can write spontaneously or from dictation, but cannot copy; word-blindness present.	6. Destruction of the visual word-centre (in some educated auditives). 7. Isolation of the visual word-centre from all afferent fibres.

The supposition is that in the large majority of cases the various lesions that have been above referred to as causes of complete or partial agraphia will be in the left hemisphere in right-handed and in the right hemisphere in left-handed persons. Of these various forms, Nos. 1, 2, and 7 should occur in all individuals alike, in association with the lesions named ; but Nos. 3 and 6 are two degrees of agraphia that may be met with in association with word-blindness, according as the individuals in whom the lesions specified occur are "visuals," or strong "auditives" much accustomed to write. On the other hand, Nos. 4 and 5 are two cases in which the same degree of agraphia may be met with, but differing by the absence of word-deafness in the one case and its presence in the other. I say *may* be, because such symptoms might not occur in a strong visual. It will be observed also that each of the partial forms of agraphia except the first is associated either with word-deafness or with word-blindness.

Defects due to functional disabilities and transitory disturbances.—In association with very various sets of conditions aphasia occurs not infrequently as a temporary disability, and sometimes with a marked tendency to recurrence, where the brief duration and the complete recovery make it improbable that it can have been due to any gross lesion.

What the proximate causative conditions have been in these different sets of cases it is mostly impossible definitely to say. Instead of softenings of brain tissue, hæmorrhages, abscesses, new growths, or traumatisms of the brain as causes of the loss of speech, we can only, in order to account for the defects of which we are now about to speak, appeal to ischæmia from irritative congestion, temporary thromboses, minute embolisms, or spasms of vessels, affecting the posterior part of the third, or the third and second frontal convolutions; in other cases to a variously brought about "inhibition," or other little understood cause leading to degradation of function in the same parts. Whatever the precise cause of the disability happens to be, this may, as with functional paralysis of limbs, persist for variable periods (hours, days, months, or even two or more years), and yet permit ultimately of a perfect restoration of function more or less rapidly brought about.

In these cases we have to suppose that from one or other of the causes above referred to, the molecular mobility of the glosso-kinæsthetic centre is so reduced, that it is unable to send down to the bulbar speech-centres incitations strong enough to call them into activity.

The principal causative or associated conditions may now be referred to in succession.

(*a*) *Irritative congestion or thrombosis, following upon excessive literary work or business worry, or as a result of exposure to cold.*—Trousseau (70) has recorded several interesting cases of this type, and one has also been published by Scoresby-Jackson. In several of these cases agraphia as well as aphasia has been present, and during recovery the patients have for a time been amnesic or paraphasic—so that the disability may have extended to the visual and the auditory word-centres, and not been confined to Broca's region.

(*b*) *Minute embolisms.*—Nothnagel (53) has called attention to cases of this kind in which patients suffering from valvular heart disease were suddenly attacked with aphasia unattended by other symptoms, the loss of speech disappearing in the course of one or two days.

(*c*) *Spasms of vessels.*—E. O. Daly has recorded a remarkable case in which "recurring attacks of transient aphasia and right hemiplegia," the duration of which varied from three minutes up to three hours, occurred in an elderly gentleman. These attacks were supposed to be due to spasms of vessels brought about under the influence of gouty and uræmic poisons in the blood. Brissaud also refers to a case of recurrent aphasia with right hemiplegia, which showed itself in a woman suffering from grave heart failure. At each relapse of the cardiac asystole there was a corresponding return of the paralysis in the right limbs with transitory aphasia.

(*d*) *Narcotic and other poisons introduced from without.*—Many cases are on record in which poisoning by stramonium, belladonna, cannabis indica, tobacco, and opium have, among other symptoms, given risen to temporary aphasia, and sometimes to verbal amnesia. References to cases of this kind, and also as to aphasia produced by snake-poison, will be found in Bateman's work (14). Aphasia has also been met with as one of the various nervous symptoms that may be produced by lead.

(*e*) *Poisons engendered within the system.*—Under this head may be included a very miscellaneous group of cases in which aphasia occurs as a temporary symptom in the course of certain specific fevers (especially typhoid, but more rarely in typhus, small-pox, measles, and yellow fever), in the puerperal state, in diabetes, Bright's disease, and gout.

In these different cases of poisoning (whether of extrinsic or of intrinsic origin) the precise mode of production of the aphasia probably varies a good deal. In some cases (i.) the altered quality of the blood may favour the occurrence of small and perhaps temporary thromboses in the vessels of the cortical centres at fault; in others (ii.) the poisons circulating in the blood may lead to contractions of the small arterioles, thus temporarily cutting off more or less completely the blood-supply of these same cortical centres; while in other cases it may be due (iii.) to the direct action of the various poisons upon the nerve elements of the cortical centres affected.

In other classes of temporary aphasia about to be referred to, the actual mode of pathogenesis would seem to be no less obscure; though in some of them it would be commonly said to be due to "exhaustion" of nerve elements following upon epileptiform discharges; or else, in still other cases, to an even more mysterious process known as "inhibition," to which some are prone to appeal where explanation is difficult.

(*f*) *Before or after epileptiform convulsions.*—It is now a familiar fact that in cases of epileptiform convulsions of the Jacksonian type, whenever the convulsion begins with twitching about the right side of the mouth (in right-handed persons), or with sensations and twitchings in the tongue, aphasia may precede and follow for a more or less brief period the occurrence of each of such fits.

A temporary aphasia may also occur in the early stages, or during the course of general paralysis of the insane, as an immediate sequence of one of the so-called "congestive attacks" to which these patients are liable.

In other cases, as we shall presently see, another form of speechlessness, known as "hysterical mutism," is found to follow some attacks of convulsions of a hysterical type.

(*g*) *In association with insanity, catalepsy, and ecstasy.*—A condition of mutism is not at all uncommon in patients suffering from chronic dementia or melancholia. It is often a lack of will to speak (sometimes under the influence of hallucinations or delusions) which leads to mutism extending, it may be, over a long series of years (64). In catalepsy and ecstasy the speechlessness is probably due to a mere suspension of volition. In all these cases, therefore, we have to do with what may be best termed "pseudo-mutism."

(*h*) *From fright and other powerful emotions.*—Many cases both of aphasia and of mutism have been recorded as resulting from these causes. In the case of an accident it often happens that the importance of the mental shock and its consequences far transcends the mere physical injury.

(*i*) *Reflex irritation in association with neuralgia, intestinal worms, etc.*—Bateman refers to three cases in which temporary loss of speech occurred in association with facial neuralgia, and also to three other cases where the presence of worms in the intestine was associated with loss of speech which disappeared more or less quickly after their expulsion (15).

(*j*) *By hypnotic suggestion.*—Charcot and others have been able to produce typical "hysterical mutism" in some hysterical patients who proved capable of being hypnotised. The mutism thus obtained during the somnambulistic period was found to persist in the waking state, unless proper steps were taken to terminate it (21).

(*k*) *Hysteria.*—The cases of temporary speechlessness that occur in hysteria belong almost solely to the type just referred to—that of "hysterical mutism." These cases are now commonly known by that name, though they were formerly included with others under the more general designation of "functional aphasia." The former name was first given by Revilliod of Geneva; the condition was subsequently made widely known by Charcot; whilst valuable series of cases have since been recorded by Cartez, Bock, and Nattier.

The view taken by Liouville and Debove to the effect that "hysterical mutism" is only a rarer and more aggravated form of a condition which is familiar enough as "hysterical aphonia" has more recently been enforced by Wyllie. This doctrine was also adopted by Charcot, who showed that there are many features common to the two conditions. Thus they occur in the same type of patients; they are produced by similar exciting causes, and are not infrequently found to alternate with one another in the same patient. Mutism is clearly the more severe condition of the two, as in it there is an absence of the power of whispering as well as of speaking aloud.

The leading peculiarities of hysterical mutism are these. Its onset is generally sudden, often after a fright or some strong emotional disturbance. Sometimes it follows a hysterical seizure, with or without paralysis of limbs. At other times it occurs without assignable cause, or it may be induced, as already stated, in some hypnotised persons by suggestion. The subjects of this disability are completely mute, presenting in this respect a notable contrast to ordinary aphasics, since they make use of no "recurring utterances" or articulate sounds of any kind. The intellect seems unimpaired, and they are able freely to express their thoughts in writing. Though the common movements of the lips, tongue, and palate are preserved, these parts are unable to act in the particular combinations needful for speech movements, and in association with the other combinations of muscular action pertaining to the vocal mechanism. Some of the muscles of the larynx are often found to be more or less paralysed, though the particular muscles involved have varied in different cases. There is frequently also more or less complete anæsthesia of the larynx and of the pharynx.

Sometimes hysterical mutism occurs in association with many hysterical stigmata, while at other times it may be almost the sole manifestation of this neurosis. Recovery may occur suddenly, perhaps after a fit or a recurrence of some strong emotion, or it may be more gradual under treatment; and in some of the latter cases there may be a sort of stammering articulation for days, or even weeks, before speech is completely restored. Several cases have been more or less rapidly cured by putting the patients under the influence of ether—during recovery from which they have begun to speak volubly. This occurred in one case recorded by Jacob, even where the dumbness had been in existence for five years (42).

It is not uncommon for mutism of this type to recur in the same individual on two or three occasions. At times, however, the attacks recur again and again over a space of eighteen months or more; this was the case in a patient who, towards the close of such a period, came under my care in the National Hospital. Delasiauve also has recorded the case of a lady who for three years was, at each menstrual period, affected with mutism and partial paraplegia. A still more remarkable case has been recorded by Hun (38), in which frequently recurring attacks of mutism were generally associated with deafness or blindness—one or both of these disabilities often ceasing after an attack of hysterical convulsions.

Much discussion has taken place as to the *pathogenesis* of "hysterical mutism." Charcot adopted the view of Marey that whispered speech is the product of the oral mechanism alone, and that the larynx takes no part in its production. He thought, therefore, that aphonia was a result of a partial paralysis of the adductor muscles of the larynx; and that hysterical mutism was due to a lack of ability to execute the proper specialised movements necessary for the articulation of words—to a partial paralysis, in fact, of the oral mechanism. Wyllie, however, following Michael Foster, maintains (72) that the larynx does take part in whispering, and that there can be no speech without the co-operation of both the oral and the laryngeal mechanisms. He believes, therefore, that disabilities of either of these mechanisms, or, of course, of both of them together, may produce mutism, and refers to various cases in Nattier's list in support of his opinion.

The question as to the part of the nervous system that we are to suppose to be at fault in cases of hysterical mutism and of aphonia now remains to be considered.

I formerly thought that these cases of mutism were instances of complete aphemia, dependent upon a functional defect in the outgoing fibres from the left third frontal gyrus (11)—because cases of subcortical defect, where there was no damage to either of the word-centres, at that time seemed to me alone capable of producing the particular combination of symptoms met with in hysterical mutism. I always recognised that it would have been more satisfactory if one could have supposed that in hysterical mutism a region of the cortex itself was at fault. It is true that in some cases of complete aphemia due to subcortical organic lesions a mutism has been produced almost exactly similar to that met with in the hysterical cases. On the other hand, a case recorded by Guido Banti has convinced me that the symptoms may not be different even when an organic lesion exists in the posterior part of the third frontal convolution, so long as it is absolutely limited thereto and leaves the cheiro-kinæsthetic centre uninjured. This very important case shows that in some individuals (besides the preservation of ability to write) the destruction of Broca's region alone may not interfere with the general intelligence any more than does a lesion in the bulbar speech-centres, or one involving the internuncial fibres between them and the left third frontal gyrus. But if such may be the case with regard to an organic lesion in Broca's region, we may all the more readily admit that a similar absence of agraphia and a similar preservation of intelligence may result from a functional disability affecting this region. We have seen that this, in fact, was Charcot's explanation of the condition (22). He considered hysterical mutism to be an instance of pure motor aphasia, resulting from a functional disability in Broca's convolution. This, however, is by no means an adequate explanation of the condition.

We have seen that Charcot, too, sharply distinguished aphonia from hysterical mutism; while we have come to the conclusion that these are, in all probability, only different degrees of the same affection—since the oral and the vocal mechanisms are probably concerned in all speech, whether it be sonorous or whispered. Still we must suppose that the cortical speech centre in Broca's region which I name the glosso-kinæsthetic is really composed of two parts—one of them being the centre for the oral mechanism, and the other, the centre for the vocal speech mechanism.

At first it was thought from the experiments of Semon and Horsley upon dumb animals that the adductors of the larynx were alone represented in the cortex, and that in the anterior part of the foot of the ascending frontal; but Risien Russell has since ascertained that the adductors are also represented in a region slightly removed therefrom. He, however, confirms the statement of Semon and Horsley that there is a complete bilateral representation of the laryngeal movements in the cortex, so that destruction of the centre on one side will not produce paralysis of the muscles of the opposite side of the larynx, seeing that they can still be called into activity by the uninjured centre in the opposite hemisphere.

If a similar bilateral representation of the laryngeal movements concerned with speech exists in man,[1] it would follow that to account for hysterical aphonia, characterised by paresis of the laryngeal adductors, we must suppose that the corresponding centres of both hemispheres have been simultaneously affected and degraded in functional activity.

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[1] This cannot, however, with certainty be considered to follow from the fact that experiments upon lower animals show that common movements of the larynx are bilaterally represented.

It has been shown, however, that hysterical mutism is intimately related to aphonia; and therefore mutism might be supposed, even for this reason alone, to be due to a bilateral affection of the cortex. But we shall now see that there is another reason pointing in the same direction.

Charcot's explanation of hysterical mutism does not account in any way for the fundamental difference that exists between it and ordinary simple aphasia. The subject of the former affection is as we have seen absolutely mute, and has no "recurring utterances" of any kind. It is commonly supposed that in ordinary aphasia due to a lesion in Broca's convolution, the recurring utterances are brought about through the instrumentality of the right hemisphere. The same kind of thing would, therefore, doubtless occur in the functional affection if the left third frontal were alone involved; but as, instead, there is absolute dumbness, we are, I think, bound to assume that in this disorder there must be a simultaneous affection of the posterior part of the right as well as of the left third frontal convolution.

Thus the clinical differences between aphasia and hysterical mutism force us to the same kind of conclusion as to the pathogenesis of the latter affection as that to which we are also led by a consideration of the intimate relations existing between it and hysterical aphonia—it compels us to believe in the existence of a bilateral cortical disability in Broca's region in each of these affections, though varying in intensity in the two cases. This explanation, I believe, has not hitherto been adduced for either of these affections.

It will be observed that in this discussion I have not adopted or hitherto referred to the hypothesis of Wyllie as to the existence of certain subdivisions of what he calls Broca's convolution. I say "what he calls Broca's convolution," because instead of limiting this region, as is customary, to the foot of the third frontal convolution, Wyllie seeks to include under it also the foot of the ascending frontal and the foot of the ascending parietal convolutions (74). He suggests that in these two latter sites the "executive cortical motor mechanisms" concerned with speech are to be found—comprising a "centre for phonation," and a "centre for the oral articulative mechanism"; while in the foot of the third frontal are stored up the "guiding psycho-motor images for spoken speech," or, in other words, what I have termed "glosso-kinæsthetic impressions." These views have been accepted and still further elaborated by Elder (32*a*), who also reports one case which he thinks "goes a long way to support the hypothesis of Wyllie."

Now, in regard to the impressions registered in the foot of the third frontal convolution, it would appear, as I have said, that Wyllie's "guiding psycho-motor images" correspond with what I term kinæsthetic impressions, and further that these impressions resulting from speech movements must partly correspond with impressions from the oral and partly from the vocal subdivision of the speech mechanism. These, according to my view, are the guiding sense-impressions which (in conjunction with auditory word-images) act directly upon, and are capable of evoking the proper activity of the bulbar-motor centres. I, therefore, do not believe in the existence of the "cortical executive motor mechanisms" for speech, which Wyllie and Elder assume to exist at the bases of the ascending frontal and parietal convolutions; and, further, I consider the reasoning which induced Wyllie to suppose that such centres might be thus situated is of itself open to very grave criticism.

He seems to have been guided altogether too much by the results obtained by experimental physiologists (Ferrier, Semon, Horsley, and others) upon monkeys and dogs, and not to have kept distinctly in mind that the movements of the larynx resulting from stimulation of the cortex in these dumb animals could not have been such movements as were concerned with speech, though they might have been the reproductions of much less specialised movements of the vocal cords (adduction and abduction) associated with respiratory acts; and again, that the movements of the lips and tongue similarly induced in these dumb animals could not afford any evidence as to the site of the "oral articulative mechanism," although they would serve to determine the seat of registration in such animals of the impressions resulting from the common movements of these parts concerned with biting, mastication, etc.

The very fact, however, that such common movements are registered in these sites (if it may be considered to hold good for the human subject) would of itself lead us to look elsewhere for the registration in man of the highly specialised movements concerned in speech—they might, for instance, be found in the contiguous foot of the third frontal convolution. And as Wyllie himself admits (*loc. cit.* p. 301)—though in a rather hesitating way—the clinical evidence points to the importance of the foot of the third frontal, and not to the bases of the ascending frontal and parietal convolutions, as the region most concerned with the production of speech. It ought to be almost superfluous to enforce the point that it is upon clinical evidence alone that we must rely, and not upon the "conclusions of the experimental physiologists" drawn from experiments upon dumb animals, for the localisation of speech-centres.

An attentive study of the case above referred to that has been recorded by Elder, seems to me to show that the lesion was an essentially subcortical one, and that all the defects in articulation met with could have been produced by damage to some of the pyramidal fibres proceeding from the foot of the third frontal convolution, even though the lesion also destroyed portions of the bases of the ascending frontal and parietal convolutions.

No real evidence is therefore forthcoming in favour of the hypothesis of Wyllie; and that the distinction which I have dwelt upon between the common movements of the lips, tongue, and palate, and the common movements of the vocal cords, as contrasted with the highly specialised combinations of movements of these parts concerned in speech is a perfectly valid one, is shown by the fact that in hysterical mutism, though the latter movements are impossible, the common movements of the lips, tongue, and palate are unaffected—just as in aphonia the vocal cords can be perfectly approximated during the act of coughing, though they cannot be brought together as a component of the movements necessary for speech.

IV. DEFECTS OF SPEECH AND WRITING DUE TO DISABILITIES IN THE AUDITORY AND THE VISUAL WORD-CENTRES

Since the publication in the year 1874 of an important memoir by Wernicke entitled *Der aphasische Symptomen-complex*, it has been the fashion to speak of the defects of speech due to lesions in the auditory and the visual word-centres as "sensory aphasia," in contradistinction to that produced by damage to Broca's region, which, in accordance with then prevalent notions, was and has since been very commonly spoken of as "motor aphasia." This mode of distinguishing these defects, though it has a certain convenience and has been widely adopted, is not in accordance with my views, as I hold the latter to be as much a sensory region of the brain as the former. It would, I believe, be much better if the term "aphasia" were restricted to the defects of speech produced by lesions in Broca's region, and the term "aphemia" to those dependent upon subcortical lesions in the course of the pyramidal fibres, leaving the speech defects produced by lesions of the convolutions around the posterior extremity of the Sylvian fissure to be grouped as so many varieties of "amnesia." Under this latter generic name many forms of speech defect would be included, due to defective recall of the auditory and the visual images of words, and produced either by lesions of the auditory and the visual word-centres themselves, or of the commissures by which they are united to one another and to corresponding centres in the opposite hemisphere. In all these cases there would be more or less interference with the recall of auditory and visual images of words. And whether we call the case one of "sensory aphasia" or of "amnesia," in each instance alike the precise degree and nature of the defect or defects would have to be settled by a systematic examination, so as to determine whether we had to do with mere diminished recollection of words, with complete loss of their auditory or visual images, or with other combinations of symptoms pointing either to partial isolation of these centres from one another, or to isolation from the general auditory or visual word-centres of which they form part.

It is true that such a nomenclature involves a slight inconsistency, seeing that aphasia and agraphia are also, in accordance with my views, forms of amnesia due to the non-revival of glosso-kinæsthetic and cheiro-kinæsthetic images respectively. But these kinæsthetic images, as I maintain, play only a small part in thinking processes, and neither of them is subject to independent conscious recall like the auditory and visual images of words which constitute our habitual thought-counters. The inconsistency is, moreover, much less than that which is entailed by speaking of "motor aphasia" and "sensory aphasia," as though the former belonged to a radically different category and really depended upon the lesion of a motor-centre. The advantage would be great of confining the term "aphasia" to its original signification, and not including under it various types of speech defects which are radically different in nature and produced by lesions in totally different cerebral regions; this is especially desirable when the objectionable generic term "sensory aphasia" can be replaced by another having a very similar general connotation.

The nature of word-deafness and word-blindness was clearly recognised by me in 1869 (12), and in 1874 Wernicke determined the region of the brain at fault in word-deafness as the hinder half of the left upper temporal convolution, with perhaps a portion of the hinder extremity of the middle temporal convolution. Wernicke was far from correct, however, in saying that the complex of symptoms resulting from such a lesion was word-deafness, paraphasia, alexia, and agraphia. This view as to the symptomatology of the lesion was founded upon too narrow a basis of observed cases with necropsies. A wider experience and knowledge necessitates its complete revision.

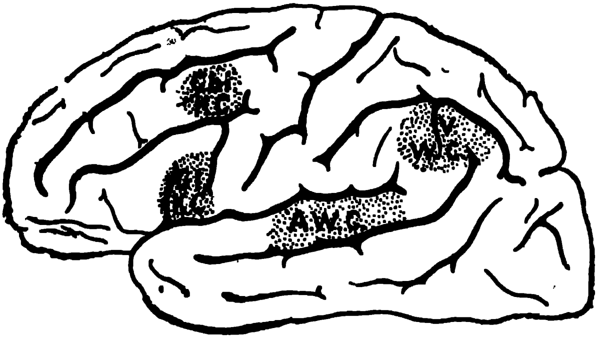

FIG. 24.—Diagram showing the approximate sites of the four word-centres in the left cerebral hemisphere.

What Wernicke originally described as a single set of symptoms named "sensory aphasia," referred by him to a lesion of the posterior extremities of the upper and middle temporal convolutions, Kussmaul shortly afterwards broke up into two groups of symptoms. According to Kussmaul, what he first termed "word-deafness" is the primary and essential result of destruction of the hinder extremities of the upper temporal convolutions; while what he termed "word-blindness" holds a similar essential relationship to destruction of the angular and parts of the supramarginal gyri. This is the view now commonly held, and both varieties are spoken of as forms of "sensory aphasia," though Wernicke's original doctrine as to the very complex results of a lesion in the auditory word-centre is held at the present day by Déjerine as well as by Mirallié (50). They both speak of word-deafness, paraphasia, alexia, and agraphia as the results of such a lesion.

It must be admitted that the functional relations of the auditory and the visual word-centres are so intimate, and their sites so close to one another, *that a lesion ~~occupying the one~~ is apt more or less to interfere* with the functions of the other for a longer or shorter period—the degree and duration of such interference varying with the nature and abruptness of the lesion, as well as with the different endowments of individuals. Disturbance to a very marked extent of the functions of the visual word-centre as a result of a lesion in the upper temporal convolutions is, however, by no means so universal as Wernicke's and Déjerine's statements as to the symptomatology of this lesion would imply. Thus, out of sixteen recorded cases of "sensory aphasia" in which the lesion was pretty closely limited to the hinder part of the first and more or less of the second temporal convolutions, in only five of them is there any mention of the existence of some amount of word-blindness.[1] Doubtless had the patients been studied more minutely (after the manner practised by Thomas and Roux), some minor amounts of word-blindness might have been found in others; but that is not what Wernicke meant, and is not other than what might be expected if we look to the close functional and topographical relations of the two word-centres.

It will subsequently be shown, moreover, that paraphasia is met with in only a little more than one-third of these cases, so that Wernicke's "Symptomen-complex" does not prove to be at all in accordance with the data at present available, although his views are still adhered to by Déjerine and Mirallié.

It must, indeed, be admitted that the defects in the auditory and the visual word-centres giving rise to word-deafness and word-blindness respectively may occur separately or together, and in the latter case the defective action in the two centres may be unequally developed. Hence the very marked clinical variations that are met with as a result of lesions in the convolutions surrounding the posterior extremity of the Sylvian fissure.

Another cause of clinical variations, even with similar lesions, is perhaps to be found in the different degrees of education of the persons attacked, and their consequent greater or less facility in reading and writing. Still another cause of variability is to be found in the varying individual endowments of patients in regard to the relative activity of their different word-centres—in other words, according as the patients are marked "auditives" or "visuals" respectively. Connate individual variations of this kind may give rise to notable clinical differences, even as results of similar lesions.

All that can be done here, therefore, is to point out the most common combination of symptoms, dealing with the various parts of the subject in the following order: (i.) defects resulting from abnormal conditions of the left auditory word-centre; (ii.) defects resulting from destruction of the auditory word-centres in each hemisphere; (iii.) defects resulting from destruction of the auditory and the visual word-centres of each hemisphere; (iv.) defects resulting from isolation of the left auditory word-centre; (v.) defects resulting from abnormal conditions of the left visual word-centre; (vi.) defects resulting from isolation of the left visual word-centre; and (vii.) defects resulting from combined lesions of the left auditory and visual word-centres, together with some remarks on the condition which has been named "psychical blindness" or "object blindness."

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[1] In my second and third Lumleian lectures particulars are given as to the cases upon which this and other following numerical statements are based.

1. Defects resulting from abnormal conditions in the left auditory word-centre.—Of all the word-centres the integrity of the auditory is of the most importance, and the defects due to different degrees of functional disability therein are the most varied, because this is the centre in which in the great majority of individuals words are first revived during thought, whether this be silent or whether it constitutes the first stage in the processes of speaking or of writing. The effects of functional degradation or of partial damage to this centre may be considered first, and afterwards those resulting from its complete destruction.

(*a*) *Effects produced by functional degradation or partial damage of the left auditory word-centre.*—In the slighter degrees of damage and functional degradation we have to do with the most typical forms of verbal amnesia, in which various words fail to be recalled as they are needed in ordinary speech. The term "amnesia verbalis" applies especially to this defect, and the objections that have been raised to it on the ground that loss of auditory images constitutes only one kind of verbal amnesia are of no practical value, because the two kinds of kinæsthetic images are, as I maintain, not spontaneously revivable as primary thought-counters, and because it is only in rare cases that visual images of words are primarily revived. The term "amnesia verbalis" is therefore especially applicable to this particular functional disability of the auditory word-centre.

This failure to recall words is always most marked with the names of persons, places, and things, these being the most specialised units of speech. The most familiar type of this defect is that which occurs as a result of defective nutrition, either with advancing years or during convalescence from prostrating diseases. Such persons are often noticed to halt in their speech, owing to their inability to recall some such words. Occasionally, however, a similar or more marked defect of the same order occurs as a result of some more or less distinct lesion of the brain.

As Lichtheim points out, evidences of amnesia are "more easy to demonstrate when the patient is made to name objects than when he is engaged in ordinary talk; names which occur without effort in fluent speaking arrest him when he has to find them for objects or persons shown to him." There is general agreement as to the fact that in amnesia words are lost in a tolerably definite order. First there are failures in the recall of proper names, then of other nouns; and only much more rarely of verbs, adjectives, and pronouns. Of this fact different explanations have been given, which cannot be discussed here.

In the slighter forms of amnesia the efforts of recollection of a person who is "at a loss for a word" tend also to call the visual word-centres into an incipient activity. Many persons, when they cannot "get out" a particular word, are often able to recollect the initial letter, and even seem to know something as to its length, as they may be able to say that it consists of about so many letters—thus seeming to show an abortive revival in the visual word-centre. But the fact that this partial revival is not associated with full consciousness of the word and does not enable it to be written, is one of considerable significance, because it seems to show how all-important in the majority of cases is the primary revival in the auditory centre, not only for the accomplishment of speech, but also for that of writing, the visual word-centre being probably called into play in writing spontaneously as well as in writing from dictation through the intermediation of the auditory word-centre.

It seems reasonably certain that in the great majority of cases in reading aloud there is first the excitation of the visual word-centre, then the passage through commissural fibres of stimuli to related portions of the auditory word-centre before the stimulus passes on to the glosso-kinæsthetic centre. This affords the explanation of another peculiarity in the class of cases of which we have been speaking, as well as of others in which the amnesia has been even more profound. Many cases have, in fact, been recorded in which the patient's speech has been so disordered that they could scarcely say more than three or four consecutive words, and could perhaps utter no nouns, in which when a book is placed before them they are capable of reading aloud almost correctly and with ordinary facility. I have seen three such cases.

Several cases of this kind are recorded in my second Lumleian lecture (7). They are, in my opinion, of great importance not only of themselves, but because of the simple explanation that may be given of them, in opposition to the theoretical view of Lichtheim, who explains them by supposing the existence of a lesion of the commissure between his imaginary "concept centre" and Broca's region. These are almost the only cases that he brings forward in support of the existence of such a centre—seeing that he gives no cases illustrating disease of the concept centre itself, and as illustrating the effects supposed to be due to damage of the other commissure in relation with the concept centre (namely, that by which the auditory word-centre is brought into relation with it), he is only able to adduce one case. But this is a case of a complicated nature, whose symptoms varied on successive days and, in my opinion, cannot be considered to give any real support to the interpretation put upon it by Lichtheim.

We have seen, then, that in slight cases of amnesia the auditory word-centre fails to respond to volitional incitations, though it may still respond to strong associational stimuli coming to it from the visual word-centre.

It sometimes happens, however, that the speech of patients is entirely limited to a mere imitative repetition of words spoken in their hearing, while they are without the power of volunteering any statement—that is, their auditory centres respond only to direct sensory incitations, and not at all to those of a volitional or associational order. In these cases, usually included under the term *echolalia*, a marked general impairment of mind almost invariably coexists.

(*b*) *Effects produced by destruction of the left auditory word-centre.*—Where, instead of partial or mere functional defects, we have to do with more or less complete destruction of the left auditory word-centre by some organic disease, a correspondingly complete word-deafness is produced, so that the patient is no longer able to comprehend spoken language. Spoken words become to such a patient mere meaningless sounds. As I have already pointed out, word-blindness (or alexia) is also an occasional consequence of such a lesion, though it is not to be regarded as a necessary accompaniment, whatever Wernicke and Déjerine may say to the contrary. These authorities, moreover, as well as Mirallié, hold that paraphasia is the kind of speech defect that is entailed by destruction of the auditory word-centre.

I formerly was of opinion that the speech defect produced by destruction of the left auditory word-centre would be aphasia (in combination with word-deafness), but a recent examination of the recorded observations in which the lesion has been limited to the hinder part of the first, and perhaps also of the second, temporal convolution (without perceptibly encroaching upon the visual word-centre) has shown surprisingly different results in regard to the nature and degree of the speech alteration met with. Thus, out of sixteen such cases contained in lists of so-called "sensory aphasia" published by Mirallié and Amidon (50), I find that what is described as "motor aphasia" existed in six of them; in six also there was some amount of paraphasia; in one case both aphasia and paraphasia are said to have existed; while in the three remaining cases voluntary speech seems to have been rather less affected.

Thus we have all been more or less wrong, and the very different results produced by destruction of the left auditory word-centre in different persons are, at first sight, not a little surprising.

A careful examination of the whole question has convinced me, however, that in postulating aphasia as a result of a destruction of the auditory word-centre, I did not sufficiently take into account the possibility of the visual word-centre being capable of acting directly upon Broca's centre, especially in the case of strong "visuals." This I now believe to be a possible cause of paraphasic speech following the lesion in question, and in some cases of even fairly correct speech. In one remarkable case recorded by Pick, fairly correct speech has even been known to occur when the auditory word-centre has been destroyed in each hemisphere (57).

Another possible cause of some amount of speech being preserved after destruction of the left auditory word-centre is apparently due to the fact that the right auditory word-centre is capable of acting upon Broca's region in the left hemisphere.

Evidence distinctly favouring both these modes of compensation will subsequently be given, and also tending to show that, when unaided by the auditory or the visual word-centres, Broca's centre alone cannot lead to the production of intelligible speech.

Thus the cases in which "motor aphasia" occurred as a result of the destruction of the left auditory word-centre are to be regarded as those in which, for one or other reason, compensatory action could not be taken on either by the left visual word-centre or by the right auditory word-centre.

2. Defects resulting from lesions of the auditory word-centre in each hemisphere.—I have found the records of four cases of this kind, and abstracts of them have been given in my third Lumleian lecture (10). In three of them (recorded respectively by Kahler and Pick, by Mills, and by Wernicke and Friedländer) speech was either unintelligible or absent, although Broca's region was uninjured in each hemisphere. But in the fourth case (that recorded by Pick) speech was scarcely at all impaired, so that its preservation drives us to the conclusion that the patient must have been a strong "visual." Words might thus have been revived in this patient in the visual word-centre, and if so incitations must have passed from it direct to Broca's region.

3. Defects resulting from destruction of the auditory and the visual word-centres in each hemisphere.—There is only one case known to me in which these combined lesions have existed in each hemisphere, although there is another well-known case, that of Laura Bridgman, in which complete blindness and deafness had been produced in early infancy by peripheral lesions, with the result that in later life she was only capable of uttering inarticulate sounds.

The case definitely belonging to this category was recorded by J. C. Shaw (68). This patient was only capable of uttering "unintelligible words" or sounds, although the third frontal convolution on each side was intact.

4. Defects resulting from isolation of the left auditory word-centre.—A clinical condition is produced by the isolation of the left auditory word-centre—that is, by the cutting it off from all its afferent fibres—which was first described by Lichtheim (47) upon the basis of a single case of word-deafness that seemed to differ from all others that had been previously recorded. To this condition Lichtheim gave the name of "isolated speech-deafness." It has been spoken of also by Wernicke as "subcortical word-deafness," whilst lately a similar condition has been described by Déjerine (who seems to have been oblivious or unaware of Lichtheim's case) under the name of "pure word-deafness." The state itself is simple enough, though an extremely interesting one. It is the condition of a partially deaf man, the deafness being not general, but limited to word-deafness. The patient can hear ordinary sounds, though more or less badly, he can talk correctly, can write correctly, can read aloud, and understand what he reads. His three disabilities are that he is unable to comprehend spoken words, and that he is consequently unable either to repeat words or to write from dictation.

I cannot agree with the pathology of the affection as given by Lichtheim or with that advanced by Déjerine. Lichtheim says (48): "Both ears are capable of receiving and transmitting excitations of speech, but intelligence of language is bound up with the activity of the left hemisphere only, so both acoustic nerves must enter into relation with the latter." Thus the affection we are considering, he adds, "can obviously come into existence only if the irradiations of both acoustic nerves in the left temporal region are broken through." He concludes that the union of these two tracts takes place in the cerebral hemisphere above the level of the internal capsule, and he believes it to occur somewhere in the white matter of the temporal lobe. A lesion in this situation, therefore, cutting off, as he assumes, the afferent fibres of both auditory nerves on their way to the left auditory word-centre, is the explanation that Lichtheim gives of the production of "isolated speech-deafness." Déjerine's view is still less satisfactory, as he speaks only of a severance of one set of afferent fibres proceeding to this centre (29).

In regard to these explanations it may be said that the view of Déjerine is certainly insufficient, since, as I have already observed, it is well known that the cutting off of the afferent impressions going to the brain from the right ear alone does not give rise to word-deafness. Lichtheim's explanation, on the other hand, is unsupported by any anatomical facts showing the distribution that he assumes to exist, which would really be that of a semi-decussation of the auditory nerves, since, after speaking of his imaginary arrangement on the left side of the brain, he adds in a footnote, "a similar distribution must obtain in the right hemisphere." Both Lichtheim and Déjerine seem to think that the right auditory word-centre is either non-existent or possessed of no functional activity, and consequently that it is not brought into relation with the left auditory word-centre. They seem also to hold similar views as to the absence of a visual word-centre in the right hemisphere. If these views were correct, surely such convolutional regions in the right hemisphere ought to show some distinct diminution in size and development as compared with those of the left side. But no one as yet seems to have noticed anything of the sort.

There was no necropsy in Lichtheim's case of "isolated speech-deafness," and from the particulars given it seems clear that the patient was generally rather deaf, altogether apart from his word-deafness. Thus my interpretation of this case some time since, after a careful study of its details, was that there probably existed pretty complete deafness on the right side, cutting off the left auditory word-centre from its proper incitations, whilst the slight sudden brain attack that is recorded to have occurred in June 1882, may in some way have destroyed the commissural fibres connecting the right with the left auditory word-centre. In this way we should have the isolation of the left auditory word-centre brought about without the necessity for postulating the existence of any hypothetical and altogether unproved distribution of the auditory fibres within the brain. We should, in fact, have to do with a man partially deaf, capable of hearing noises only, but not able to perceive the meaning of spoken words, owing on the one hand to the complete isolation of the left auditory word-centre from all sounds, and on the other to the inadequacy for this purpose of the right centre alone, because of the imperfect development of associational fibres in connection with speech functions in this hemisphere. This view has since been rather confirmed by the record of another case by Sérieux, in which it is definitely stated that the patient was absolutely deaf on the right side as a sequence of an old otitis (66).

Strangely enough it has been found that a very similar clinical combination may be produced in quite a different way, as may be seen by reference to the very remarkable case recorded by Pick, and previously referred to, in which there was destruction of the auditory word-centre in each hemisphere. Here the clinical condition was almost identically the same. There was even a certain amount of general deafness, though it was not due to middle-ear disease, nor did it seem to be definitely localised to one side. As I have already said, this case seems absolutely inexplicable unless we are to suppose that the patient was a "visual" who could speak, write, read aloud, and understand what he read, mainly through the activity of the visual and the kinæsthetic word-centres—at all events without the co-operation of the auditory word-centres, seeing that these were destroyed. The case shows, moreover, that general deafness does not result from destruction of these special word-centres even in both hemispheres. The patient's general hearing power was defective, though far from lost.

These are the only three cases of "isolated word-deafness" known to me.[1] It will be seen from what I have said that this rare group of symptoms is capable of being induced in two very different modes—just as we shall find subsequently that what Déjerine has called "pure word-blindness" may also be produced in two different modes, and that the modes are analogous in the two cases. Pick was fully aware of the very unusual nature of his case. He recognised that it could not be a case of subcortical word-deafness of Lichtheim's and Déjerine's type, though he was not prepared to advance any other interpretation. Mirallié (51), however, has been so very uncritical as to cite it as an example of Déjerine's type, notwithstanding the fact of the existence of the double cortical lesions occupying the sites of the auditory word-centres.

It should be added that in word-deafness generally it often happens that the patient can recognise his own name, just as the word-blind patient may be able to write his name and nothing else. This most familiar word of all is the one the recognition of which persists the longest, and after the meaning of all other words may have been blotted out. In other cases the word-deafness is not complete. There may be still the power of comprehending many familiar words, and by this means patients occasionally may guess correctly what is being said to them. They may, therefore, for the moment appear to understand more than they really do, as the questioner may ascertain by using some of the same words, but in different combinations, and so as to require different answers. For reasons of this kind some of the slighter forms of word-deafness may escape observation unless specially looked for. Again, where the word-deaf person has been acquainted with two or more languages, that which is most used continues to be understood the longest—just as, during recovery, it is the first to be again comprehended.

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[1] Though Wernicke says a few words concerning a case that had come under his observation (see *Fortschritte der Medicin*, 1886, p. 474).

5. Defects resulting from abnormal conditions of the left visual word-centre.—There is not the same variety in the defects of speech caused by disease of the visual word-centre that is to be met with as a result of disabilities in the auditory word-centre. This is apparently due to the fact that in the great majority of persons voluntary revival of words occurs primarily in the auditory word-centre, while the visual word-centre is called into activity mainly by stronger stimuli—either by those coming to it through associational fibres or by still stronger incitations that reach it directly from without. It is only in comparatively rare cases, as we have seen, that the visual word-centre takes the place occupied in the great majority of individuals by the auditory word-centre, as the seat in which words are primarily revived in silent or in spoken thought. Consequently the speech defect known as "amnesia verbalis" occurs almost only as a result of a lowered functional activity in the auditory word-centre, and only in extreme rare cases (where the individual is a very strong "visual") as a result of mere lowered activity in the visual word-centre. Since a volitional stimulus is weaker than that which comes through an associational channel from a centre strongly aroused, and weaker still than that which comes to the centre directly from without, and since a lowered nutrition or diminished molecular mobility of a centre from any cause might lead only to a failure in the centre to respond to the weakest stimuli, it is to be expected that such results in relation to language would show themselves only in connection with the centre accustomed to respond to such stimuli (namely, the auditory word-centre), and would be almost absent in connection with the visual word-centre.

Consequently we know next to nothing about the effects of mere functional degradation in the visual word-centre, that is, of slight disabilities such as when occurring in the auditory word-centre are productive of the various degrees of amnesia verbalis already described. All that can be said is that when such functional degradation of the visual word-centre is present it would, (*a*) in the rare event of the patient being an extremely strong "visual," of itself tend to produce amnesia verbalis, as was seen in a patient under the care of Charcot, whose case is referred to by Ballet (2); or (*b*) even in an "auditive" tend greatly to aggravate any disability that may have been caused by a coexisting defect in the auditory word-centre, and also to hinder recovery therefrom; and further (*c*) where the functional defect involves both word-centres the amnesia ought to be more than usually bad and probably associated with more or less of paraphasia.

Effects produced by the destruction of the left visual word-centre.—We have to pass, then, at once to a consideration of the effects produced by destruction of the left visual word-centre, which is now generally supposed to be situated in the angular and possibly in part of the supramarginal convolution. These effects will be found to vary in different individuals, just as variation was found to be the case when we had to do with the results of lesions in the left auditory word-centre.

One important difference between the effect of lesions of the visual word-centre as compared with those following upon lesions of the auditory is that speech is very little, if at all, interfered with. There is no well-marked aphasia or paraphasia, only a very slight amount of paraphasia in some of the cases.[1] That there should at times be slight disturbances of speech is not to be wondered at if we bear in mind the close functional relationships of the visual and the auditory word-centres, and how easily therefore the functions of the latter may be disturbed by a lesion in the former.

It is often found that word-blindness is associated with right-sided homonymous hemianopsia—that is, on the same side as paresis of the limbs—for there is often no complete hemiplegia in these cases. The hemianopsia is generally due to destruction of the "optic radiations" of Gratiolet; but where the lesion is quite limited to the cortex these fibres will not be involved, so that hemianopsia is not a necessary accompaniment of word-blindness.

Agraphia also is a symptom sometimes present and sometimes absent in cases of word-blindness. The presence or absence of this symptom is, however, of much more fundamental importance than the presence or absence of hemianopsia. It is best, in fact, to divide cases of word-blindness into two categories—namely, (*a*) cases in which the word-blindness is associated with agraphia and (*b*) cases in which there is no agraphia. In the latter group it is found that the individuals can write as well with the eyes closed as when they are open, and further that they are, even after a brief interval, unable to read what they themselves have written.

(*a*) In the cases where agraphia is present it is commonly found that such patients are capable of writing their names correctly, although they can write nothing else. The signature is an emblem which may, by reason of its familiarity, be executed by the cheiro-kinæsthetic centre with the smallest amount of prompting; and it would appear that this prompting may come from the common visual centre in cases where the left visual word-centre is destroyed. Similarly a word-blind patient may be able to recognise his own name when he sees it, though apart from this there may be complete alexia. As Déjerine says: "He recognises it by its general form, by its physiognomy, and not by the assemblage of letters of which it is composed,"—just, in fact, as he would recognise a geometrical figure or any other drawing. Differences of degree are, however, met with in word-blindness. In some cases, though the patients are unable to recognise single words (word-blindness), they can recognise individual letters; whilst in others not even letters can be recognised (letter-blindness). These two forms represent differences in kind rather than of degree, all intermediate forms may be met with. Sometimes patients will recognise certain letters and not others; and in one case recorded by Batterham vowels only were recognised.

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[1] For instance, in Nos. 44, 45, 47, 49, 50, and 52, Mirallié's list of cases included under the heading "Cécité Verbale" (pp. 152-157).

Déjerine and Sérieux have each of them published typical cases of this type in which word-blindness was associated with agraphia, and where the lesion was strictly limited to the visual word-centre. In the case recorded by the former observer, which occurred in a day-labourer to whom writing was a comparatively unfamiliar exercise, the agraphia was absolute (26). Damage to the visual word-centre under such circumstances might be expected to produce its maximum results in this direction. In this case also there was hemianopsia, while in that of Sérieux it was absent (67).

(*b*) In the second group of cases word-blindness is not associated with agraphia, the individuals being able to write, and as well with the eyes closed as open. (As I have already said, a precisely similar group of symptoms may be produced in a totally different manner, quite independently of destruction of the left visual word-centre. These cases will be fully considered in the next section.)

The effects of destruction of the left visual word-centre are liable to vary much in different individuals in accordance with their different sensorial aptitudes and different degrees of education, just as we have found the results of destruction of the left auditory word-centre to present marked differences in different cases. The results already described under group (*a*) are those which most frequently follow when the left visual word-centre is destroyed in ordinary individuals. But suppose a similar lesion to occur in a person who is a strong "auditive" though a weak "visual," and at the same time, perhaps, an educated person who has previously been in the habit of writing much. It may happen in such a person, after the stage of learning to write has well passed, that the activity of the visual word-centre may be reduced to a minimum during the execution of such acts. We have seen reason to believe that the words about to be written become nascent first in the auditory word-centre, and it seems fairly probable that in individuals with the endowments above mentioned this centre, rather than the visual word-centre, may be the one which acts upon, and co-operates with, the cheiro-kinæsthetic centre, just as we have seen that in certain persons who are strong "visuals" the visual word-centre may be capable of co-operating directly with Broca's centre for the production of articulate speech. I contend, therefore, that the preservation of ability to write in cases where there has been word-blindness, and where the visual word-centre has subsequently been found to be destroyed, is to be explained in this way—that is, by supposing that the auditory word-centre, instead of acting, as it usually does, by rousing the visual word-centre to conjoint action, in these cases acts directly upon the cheiro-kinæsthetic centre (by way of the commissure *ff*, in Fig. 23).[1] The writing thus produced may be fairly good and without mistakes, though that of other patients may show defects of a paragraphic type. The handwriting itself, too, is usually larger than that which was previously customary to the patient. There is also the peculiarity that these patients can write as well with the eyes closed as when they are open, and the still further peculiarity that they cannot subsequently read what they have written except by a manœuvre which causes a stimulus to pass in the reverse way—that is, from the cheiro-kinæsthetic centre back to the auditory word-centre (in the direction *f′f′*, Fig. 23). This is brought about by passing the tip of the finger over the outlines of the letters and so reading off the result. The possibility of this mode of reading by the "tip of the finger" seems to have been first noticed in a word-blind patient by Westphal (72).

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[1] It is worthy of note that both Kussmaul and Spamer seem to think that this is the ordinary way in which writing is effected (see Kussmaul, *loc. cit.* p. 777).

There are only two cases with necropsies to which I can refer in illustration of this group, and unfortunately they are not very well-defined cases, being rather complicated, not only clinically, but also by reason of the lesions found at the necropsy. The first of them is also, I believe, the earliest recorded instance of any such defect. It is a remarkable case that was published long ago by Broadbent. The other case is one that has been recorded by Osler. Both of them are quoted in my third Lumleian lecture (8).

It will be found that in each of these cases in which word-blindness has been present without agraphia the patients have been more or less educated persons well accustomed to write, and that the incitations which lead to this act could not have come during their illness from the damaged visual word-centre. Therefore it seems only open to us to suppose that these patients continued able to write spontaneously or from dictation, simply because they were able to call up the familiar activity of the cheiro-kinæsthetic centre directly at the instigation of the auditory word-centre. It is well known that many voluntary movements during the period that they are being acquired, and sometimes for long afterwards, require for their execution the active co-operation of the visual centre, though at a later period the guidance of the kinæsthetic centres may suffice for their production.

6. Defects resulting from isolation of the left visual word-centre.—Isolation of the visual word-centre is a term employed here in the same sense that isolation of the auditory word-centre was previously spoken of; it is not a complete isolation, but merely a cutting of the centre off from all its own afferent fibres, as well as from all communication with the corresponding centre of the opposite hemisphere.

Seeing that each of these clinical groups of symptoms may be produced in two distinct modes—that is, either by cortical or subcortical lesions, it seems best to adhere to Déjerine's nomenclature and speak of them as "pure word-deafness" and "pure word-blindness" respectively.

The cases of pure word-blindness with which we are now concerned, in which the visual word-centre remains intact, are comparatively few in number. Three complete cases with necropsies have been recorded (by

Déjerine, Wyllie, and Redlich), while of two others the clinical details only have been published. Déjerine's case is remarkable for its completeness and the great care with which it has been recorded (30). Its most notable features were the absence of any hemiplegia; the combination of complete right lateral hemiachromatopsia with partial right hemianopsia; the absence of word-deafness or speech defect of any kind; the absolute word-blindness coupled with ability to write freely both spontaneously and from dictation, whilst ability to copy writing was very defective; and the length of time that these symptoms lasted without change. The subsequent sudden supervention of complete agraphia associated with paraphasic speech, followed by the discovery of a recent lesion in the angular gyrus and adjacent parts, were also very significant features of the case.

In each of the other two cases (for abstracts of which see *Lancet*, May 1, 1897, p. 1190) there was also no motor paralysis, only a very slight amount of right-sided hemianæsthesia. There was right hemianopsia in each, but no mention is made of hemiachromatopsia in the cases of Wyllie and Redlich, or of the ability to read words by means of kinæsthetic impressions, though both were present in the case of Déjerine. It seems highly probable, however, that one if not both of these characteristics might have been met with had they been specially looked for. They both existed in one of the two incomplete cases before referred to—namely, in one observed by Gaucher, and recorded by Mirallié (52); while in the second of these cases, recorded by Batterham (16), though there is no mention of hemiachromatopsia, the ability to read words by aid of kinæsthetic impressions was present. Word-blindness was not complete in this case, and Batterham says: "When asked to spell out a word written in the 'round hand' of the copy-books, she failed with several letters; but on being told to copy the unrecognised signs, or to run her pencil over them as if writing them, she in most cases recognised their name and significance. This experiment was repeated several times, and the patient was delighted to find that she could 'jog her memory' of letters in this way." Both these cases are well reported, and are worthy of careful study, although there is no record as to the pathological causes of the clinical conditions.

The lesions found in each of the three cases where a necropsy was made have shown a striking similarity. In each there was softening and atrophy of the white substance of the occipital lobe, together with more or less damage to certain convolutions—that is, to the lingual and fusiform lobules as well as the hippocampal gyrus and the cuneus, or, speaking more generally, to some of the convolutions on the under and inner surface of the occipital lobe. It seems quite probable, however, that the lesions of the convolutions may be of little significance so long as there is the presence of extensive destruction of the white substance of the occipital lobe. Nothing more definite can be said on this subject at present. Déjerine attaches most importance to the destruction of portions of the white matter—namely, of that in which would be included (*a*) the "optic radiations" of Gratiolet (*b*), the fibres proceeding from the left half-vision centre to the left visual word-centre, as well as (*c*) the fibres from the right half-vision centre to this same word-centre. This he indicates in a diagrammatic figure of some complexity.

It is clear from the explanation that Déjerine advances of his case (31), and also from what he says elsewhere, that he does not believe in the existence of auditory and visual word-centres in the right hemisphere in ordinary right-handed persons. On this subject he takes much the same view as Lichtheim, and now explains "pure word-blindness" in a fashion analogous to that by which the latter explained "pure word-deafness"—namely, by supposing the severance from the left visual word-centre of the associational fibres connecting it with the general visual centre in each hemisphere (see Fig. 25).

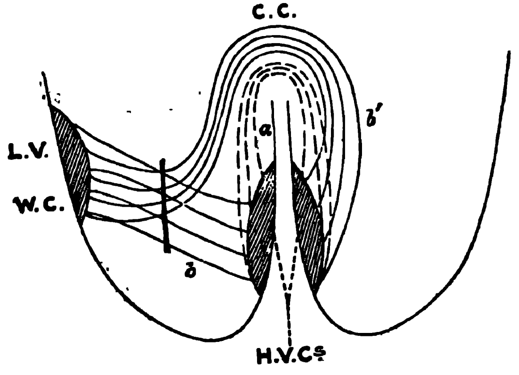

FIG. 25.—A simplified diagram representing Déjerine's view as to the mode of production of pure word-blindness. H. V. C., Half-vision centres; L. V. W. C., left visual word-centre; C. C., posterior extremity of corpus callosum, containing commissural fibres (*a*) connecting the half-vision centres and also fibres (*b'*) from the right half-vision centre to the left visual word-centre. (The "optic radiations" have been omitted.) The dark line indicates the site of a lesion which would cut off the left visual word-centre from the half-vision centre of each side.

I, however, believe in the existence of a visual word-centre in each hemisphere (though unequally developed), and that the two are brought into functional relation with one another by means of commissural fibres in the posterior part of the corpus callosum. I believe also that each of these visual word-centres would be in relation by other associational fibres with the half-vision centre of its own side (which both of us suppose to be commissurally connected with its fellow); and that Déjerine's form of pure word-blindness may be produced by severance of the associational fibres between the left visual word-centre and its corresponding half-vision centre, together with a lesion of the commissure between the two visual word-centres, in some part of its course (see Fig. 26). The isolation of the left visual word-centre would thus be complete; and brought about, moreover, in the same sort of way that I have postulated for the isolation of the auditory word-centre in the condition that Déjerine calls "pure word-deafness."

The commissural fibres between the two word-centres may be damaged, in accordance with my interpretation, by the lesion in the white substance of the occipital lobe extending far enough forwards to involve them, just as Déjerine supposes those from the right half-vision centre to have been destroyed; or else by a separate lesion in the posterior part of the corpus callosum, such as was actually found in Déjerine's case, or in the part of it known as the tapetum.[1]

When we consider how one eye suffices for perfect vision, although only one-half of the proper amount of visual fibres goes to each half-vision centre, we may see all the more fully how complete and intimate must be the coactivity of these centres in vision, even when one eye only exists. That being so, it would be in vain with our present imperfect knowledge to attempt to define the actual mode of association of the half-vision centres with their outlying portions which we name visual word-centres. All I can say is that such an arrangement as I have previously indicated seems to me to be much more in accordance with all known probabilities than that of Déjerine, who assumes that the right angular gyrus has practically no visual functions, just as he assumes that the posterior half of the right upper temporal convolution has no auditory functions. What I have previously said as to the partial co-education of the centres in each hemisphere, as well as what we know as to the modes in which recovery takes place in various forms of speech defect, may be considered to lend support to my view.

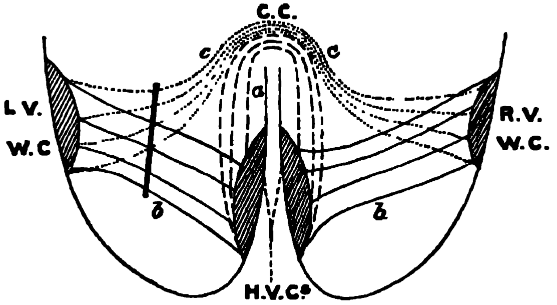

FIG. 26.—A diagram representing my view as to the mode of production of pure word-blindness. C. C., Posterior extremity of corpus callosum; *c*, *c*, commissural fibres connecting the two visual word-centres; *b*, *b*, fibres connecting each half-vision centre with the visual word-centre of the same side.

As I have already indicated, the two modes of producing pure word-blindness are pretty strictly comparable, *cæteris paribus*, with two modes of production of pure word-deafness. One of these forms, which for the sake of distinction may be termed the parietal type of pure word-blindness, is brought about by destruction of the left visual word-centre in persons by whom writing can be executed under the instigation and guidance of the left auditory word-centre; while the other (Déjerine's form), which might be called the occipital type, is due to isolation of the left visual word-centre from the half-vision centre of its own side as well as from the opposite visual word-centre.

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[1] The tapetum may perhaps include the commissural fibres between the two visual word-centres, and it is specially stated in Redlich's case that these fibres were in part degenerated, as also was the forceps major, which probably includes the fibres connecting the two half-vision centres with one another.

At present there seems no very definite means of diagnosing these two types of pure word-blindness from one another during life. The following considerations may, however, afford some help:—

Parietal Type of Pure Word-blindness.	*Occipital Type of Pure Word-blindness.*
Possibly some right-sided paresis.	Probably no right-sided paresis, but possibly some slight right hemianæsthesia.
Speech often slightly paraphasic.	Speech not affected, or slightly amnesic only.
May be no hemianopsia or hemichromotopsia.	Hemianopsia always, and when incomplete probably also hemiachromatopsia.

These seem to me to be the only approximations to differential characteristics that can be suggested at present, some of them being based upon the fact that destruction of the visual word-centre may be associated with a lesion in the parietal region, and consequently may be associated with some amount of right-sided hemiparesis; while in the cases of occipital type, if the lesion of the occipital lobe extends sufficiently far forwards, we may get more or less marked hemianæsthesia though without any motor paralysis. The association of alexia without agraphia in a person whose speech is not appreciably interfered with, and who has slight hemianæsthesia without paralysis, would, in fact, afford strong presumptive evidence that the pure word-blindness was of the occipital type.

7. Defects resulting from combined lesions in the left auditory and visual word-centres.—It will have been seen from the cases already recorded that destruction of the left auditory word-centre or of the left visual word-centre alone, and especially in the last case, may be quite compatible with the preservation of a fair amount of intelligence. The result is, however, altogether different when both these centres are badly damaged at the same time. The unfortunate individuals thus affected are often reduced to a most deplorable condition, seeing that they can usually neither speak nor write intelligibly, and that they are unable to understand the speech which they hear or the language they may see either in writing or in print. They can mostly communicate with others, and be communicated with, only by means of signs and gestures; and at the same time they must necessarily suffer a very distinct amount of mental impairment, owing to the blotting out of the principal linguistic symbols by means of which all but their most elementary thinking processes are carried on.

Although this is the kind of condition that has been met with in the majority of these cases, yet a careful examination of all the published records shows that, as in cases where there is destruction of the left auditory word-centre, so here with combined lesions of the auditory and the visual word-centres, a considerable variation is met with in different cases. These variations depend partly upon differences in the relative completeness of the lesion in one or other of these centres; and in cases where one of the centres is incompletely destroyed probably also to a considerable extent upon individual variations in original endowment—that is, upon the question whether the patients are "auditives" or "visuals"—as well as in part upon their degree of education. Where the destruction of both the left word-centres has been complete, moreover, variations in symptoms may depend upon the degree of development of the corresponding centres in the opposite hemisphere.

Taking fifteen of the most typical cases I could find on record, in three of them the lesion was complete in the visual and partial in the auditory word-centre; in two it was complete in the auditory and partial in the visual word-centres; in four cases it was incomplete in both word-centres; while in six cases the lesion was pretty complete and equal in both word-centres.

Looked at from another point of view, that is, as to the kind of speech defect presented by these fifteen cases, I find that speech was more or less good in two; that there was more or less marked paraphasia or actual jargon-speech in seven, and more or less complete speechlessness in six. These different results may at first seem very astonishing to others as they did to me. But the more one thinks of cases presenting this double lesion, in which speech was only slightly paraphasic or fairly good, the more it seems necessary to suppose that the auditory word-centre of the right hemisphere must have been able to act upon and with the left glosso-kinæsthetic centre. These cases cannot be explained by supposing that speech was produced by the right auditory word-centre acting with the right glosso-kinæsthetic centre, because all present knowledge goes to show that this could only be brought about after a long interval, during which these centres were educated to act together. In the cases in question, on the other hand, there was evidently no such interval, seeing that the modified speech was initiated in each case just after the brain lesion occurred. It is worthy of note also that in the two cases in which speech was best preserved the visual word-centre was only partially damaged, so that some help may also have come from its co-operation.

A case recorded by myself belonging to this group is one of a very extraordinary nature in many respects, the patient having lived over eighteen years after his seizure, and his speech defects having remained constant throughout almost the whole of this period. His spontaneous speech was limited to a few words, and, though he was neither word-deaf nor word-blind, both the auditory and the visual word-centres of the left hemisphere were completely destroyed. The record of this remarkable case, together with illustrations of the brain, will be published in the forthcoming volume of the *Transactions of the Royal Medical and Chirurgical Society* for 1897.

Object-blindness.—In association with the latter class of cases, and of others in which the lesion extends into the occipital lobes, another defect is sometimes met with which has been commonly known as "mind-blindness," but which is, I think, much better termed "object-blindness" (Ballet and Wyllie). This latter term is not only more descriptive of the nature of the defect, it is also a better companion term for "word-blindness." The two conditions are very frequently associated, and when the former is present, although the patient sees relatives or other familiar persons, he may not recognise them. Similarly he seems unable to recognise and to be unaware of the uses of common objects.

This effect is probably due to a partial isolation of the common visual centre—the cutting across not of the afferent fibres, but of the fibres by which the centre is brought into association with other sensory centres. A failure in the irradiation of associational impressions which are wont to pass from the visual to other sensory centres on the presentation of an object to the visual sense would, of course, absolutely check the neural processes essential to perception, and thus lead to the non-recognition of the object and its uses. An excellent example of this condition has been recorded by Bernheim.

Optic aphasia is a term that has been given, rather needlessly I think, by Freund to a condition in which, though the person quite well recognises objects or persons, he is unable to call up the name from the visual impression only, but may be able to do so at once when the visual is reinforced by some other sensory impression—either by touching the object, smelling it, or tasting it—but especially by touching it. This form of inability to name at sight may be brought about in various ways:—(*a*) by severance of the associational fibres between the common visual centre and the visual word-centre; (*b*) where the visual word-centre is weak, it may then respond to two incitations though not to the visual alone; (*c*) when the visuo-auditory commissure is damaged in a person who is not a strong "visual."

Inability to read or write music, or to read and write numerals.—It is often found that the patient's inability to read or write music or numerals is not at all proportionate to his inability to read or write words.

In reference to music, a case was long ago recorded by Lasègue in which a musician completely aphasic, and unable to read or write in the ordinary way, could after hearing a passage of music write such passage on paper with facility. Many other instances of the same kind have since been recorded; the inference from which would seem to be that the seat of registration of the two kinds of impressions may be more or less separate, though in all probability contiguous.

At other times there may be complete word-blindness and musical-blindness, but with preservation of ability to read and write figures and to calculate, as in the celebrated case of "pure word-blindness" recorded by Déjerine. It is, indeed, very frequently found that there is this ability to read and write figures with varying degrees of facility, when the reading or writing of letters or words is impossible. This curious fact has given rise to much discussion and to many attempts to explain it. It seems to me that various reasons are probably operative, though in different combinations in different cases. Some of the most important causative conditions may be these:—(i.) the greater simplicity of the recollection of numerals as compared with letters—there being only nine of the former as against twenty-six of the latter; (ii.) there is the fact that our attention is much more habitually called to individual figures, while letters tend to be merged into multitudinous words; (iii.) there is the possibility that numerals may much oftener than words be primarily recalled in the visual rather than in the auditory word-centre; and (iv.) the further possibility that numerals may be registered in some region of the general visual centre apart from the visual word-centre.

Paraphasia and **paragraphia.**—Paraphasia and paragraphia are incoördinate rather than paretic or paralytic defects of speech. We may describe each of these defects as occurring under three grades or degrees of severity, though the grades of each are often dissimilar as they exist in the same persons. In the minor form of the defect (i.) the patient uses some wrong words instead of those that he intends to employ. In a more severe form (ii.) he collocates words in such a disorderly manner as to convey no definite meaning. In the most severe form (iii.) the patient does not make use of words at all, but utters a mere gibberish or jargon in which actual words are not to be detected; or in writing makes random collocations of letters, or perhaps mere unmeaning strokes not even representing letters.

The first variety is the most common and is generally associated with a certain amount of amnesia, when the patient will often substitute some noun of general import (such as the word "thing") in place of that which he is unable to recall, or else he substitutes a periphrasis in order to express his meaning. An allied defect may also be met with in the form of a transposition of syllables of words in the same sentence, or the interposition of letters that do not belong to the word—a condition to which Kussmaul gave the name of "syllabic stumbling." Defects in writing of a similar kind are also met with.

All transitions may be encountered between the first and second grades of paraphasia, and they are probably similar in nature and mode of production. Some of the slighter defects are apt to show themselves in weak and exhausted states of the system, and in cases where there is impaired nutrition of the brain, either from previous disease or from old age. They may occur also when the attention of the person speaking is distracted by counter currents of thought being carried on at the same time. In all such cases wrong words are particularly apt to slip out.

Apart from these causes of a more general order what has already been said in previous sections seems to show that either paraphasia or paragraphia, or sometimes both in the same patient, may be produced by a lesion which causes speech or writing to be performed by some unaccustomed cerebral process. Paraphasia not infrequently occurs, for instance, in a case of word-deafness when the visual has to take the place of the auditory word-centre in the incitation of Broca's region; or in cases of combined word-deafness and word-blindness when the right auditory word-centre has to take on the functions of the left. In each of such cases there is very apt to be, at all events for a time, either a want of proper association between the new centre in which words have to be revived and the several sensory centres and their annexes, or else an incoördinate action between the driving centre and the centre driven.

Under similar conditions paragraphia may also show itself, because here too there would in one case be a change in the locus of the initiatory recall of words and a less perfect system of associational fibres radiating therefrom, as well as a less evolved system of associational channels between this newly dominant site for the initiation of words and the left cheiro-kinæsthetic centre. Paragraphia may also be caused in cases where the visual word-centre is destroyed in a person much accustomed to write, when this act begins to be brought about (as it seems to be in some cases) by the auditory word-centre acting directly upon the cheiro-kinæsthetic centre.

In addition to the causes already mentioned some partial damage to the auditory word-centre may possibly cause paraphasia; just as partial damage to the visual word-centre may possibly cause paragraphia.

Wyllie seems to suppose that in cases of word-deafness the paraphasia which often occurs may result from an unguided action on the part of Broca's centre (73). Thus, he says: "In cases of word-deafness—which implies the obliteration of the auditory store of images—the motor-centre may emit the wrong words when the patient tries to speak." As I have shown, however, in previous sections, Broca's centre alone appears to have no power of initiating intelligible speech—undirected it seems only to lead to the production of mere gibberish.

Lichtheim's view, again, that paraphasia is dependent upon a severance of the associational fibres between the left auditory and glosso-kinæsthetic centres (his so-called "commissural aphasia") is founded upon no sufficient evidence, as Déjerine (28) has also pointed out. Destruction of the auditory word-centre in a certain number of cases leads, as I have shown, to paraphasia, and in about an equal number it leads to aphasia. Precisely the same thing, therefore, must hold good in regard to severance of the associational fibres between the auditory word-centre and Broca's centre. That is, paraphasia might be produced when either the left visual word-centre or the right auditory word-centre were capable of partially taking on the functions of the left auditory word-centre when the latter has been cut off from its connections with Broca's centre; while aphasia would result in cases where from one or other cause neither of the centres mentioned could take on compensatory functions, so as to incite and direct the action of Broca's centre.

In the third form of incoördinate amnesia speech is reduced to a mere jabber of meaningless sounds, and writing to a mere scrawl in which words and sometimes individual letters are indistinguishable. It is often found that these two disabilities coexist, and that the person so affected is also word-deaf and word-blind, and consequently does not know that what he says or writes is mere gibberish. This is what happens in certain cases of destruction of the left auditory and visual word-centres when the corresponding centres in the right hemisphere remain comparatively undeveloped, and still more certainly when each hemisphere is the seat of this double lesion. Again, much the same condition may exist (save for the possible absence of word-blindness) in cases where both auditory word-centres are destroyed.

An extreme defect in speech of jargon type, or a similarly extreme defect in writing in certain cases, however, exists alone—that is without the association either of word-deafness, word-blindness, or mental failure. These cases, therefore, belong to a totally different category from those last referred to.

A well-known instance of such a defect in speech, without defect in writing, was recorded long ago by Dr. Osborne. It occurred in a scholar of Trinity College, Dublin, who was able to talk only in a meaningless jargon and read aloud in much the same way, though he perfectly comprehended what he read, and all that was said to him, and expressed his ideas in writing with considerable fluency. His power of repeating words after another person was, however, confined to certain monosyllables. This case seems to me only explicable on the supposition that it was due to some perverted activity in the glosso-kinæsthetic centre.

In other cases the defective power of expressing occurs in writing rather than in speech. Hughlings Jackson (40) and P. J. Cremen have each recorded one such case, and I have recorded another. Though these cases were by no means free from other complications, there seems reason for believing that the agraphic defect must have been especially due to a perverted activity in the cheiro-kinæsthetic centre.

The case recorded by myself (13) may, perhaps, have been of a somewhat different type. There was a constant repetition in this patient's writing of certain groups of letters, which varied from time to time; and the case also afforded an admirable example of what Gairdner long ago termed "intoxication of the brain" with a word or letters, and of which he gave various examples. A similar intoxication with words is not infrequently shown by aphasics, or by others who are partly agraphic. In all such cases, however, we may expect to meet with evidence of some general mental defect over and above that which may be due to the mere lesion causing aphasia or agraphia.

V. DEFECTS OF SPEECH AND WRITING DUE TO DAMAGE TO THE COMMISSURES BETWEEN THE DIFFERENT WORD-CENTRES

It seems better to reserve, as I have for some years done, the word "commissure" as an appellation for the fibres which connect centres of like kind, that is, either sensory centres or motor centres; and to name "internuncial" the fibres which connect sensory with motor centres. These two orders of fibres will thus be clearly distinguished from one another, as well as from so-called sensory and motor nerve-fibres—namely, those that connect the periphery with sensory centres, or those that connect motor-centres in the bulb or cord with muscles.

The commissures with which we are now principally concerned are (i.) those between the auditory and the visual word-centre; (ii.) that between the auditory word-centre and Broca's centre; and (iii.) that between the visual word-centre and the cheiro-kinæsthetic centre. We shall briefly refer to them in this order, and in all cases we must be supposed to have to do with structural defects in the course of these fibres, or else with some unnatural pressure upon them.

(i.) *Defects due to damage to the two commissures between the auditory and the visual word-centres.*—One of these commissures conducts impressions from the auditory to the visual word-centre (the *audito-visual commissure*) and is called into play in writing from dictation, and probably also in any spontaneous writing. The other conducts impressions from the visual to the auditory word-centre (the *visuo-auditory commissure*), and is habitually called into play when we read aloud or name an object at sight.

Where both of these commissures are destroyed in the same person without other notable damage a very interesting group of symptoms is produced. The patient understands perfectly all that is said to him and what he reads. He cannot read aloud a single word or letter, though immediately that he hears the word or letter pronounced he can at once repeat it. Similarly, he cannot write a word or letter from dictation, but he can at once copy any such word or letter that he has before him. Spontaneous writing is similarly impossible, though if the auditory centre be intact spontaneous speech may not be very much interfered with. An interesting case that was for very many years under my observation presenting a group of symptoms of this kind will be found recorded in the *Medico-Chirurgical Transactions* for 1897.

Cases which I interpret as illustrating defects of the audito-visual commissure alone have been published by Pitres (61), who, however, brings them forward as instances of destruction of the cheiro-kinæsthetic centre. Another such case has also been recorded by Dingley.

(ii.) *Defects due to damage to the commissure between the auditory word-centre and Broca's centre.*—To the result of such a lesion (the "commissural aphasia" of Wernicke) I have recently referred when speaking of paraphasia, so that nothing more need be said here.

(iii.) *Defect due to damage of the commissure between the visual word-centre and the cheiro-kinæsthetic centre.*—Such a lesion should in most persons produce complete agraphia—nothing more than slow and tedious copying being possible (as a drawing would be copied) under the guidance of the general visual centre. But in persons who have been much accustomed to write it is possible that writing (though at first of a paragraphic type) may be executed, owing to the auditory word-centre acting directly upon the cheiro-kinæsthetic centre.

The other commissures seem to be only for occasional or supplementary use, and the effect due to their severance (tending to check compensation in different ways) may be easily imagined by reference to the centres which they connect, together with the direction in which the stimulus passes.

Thus, referring to Fig. 23, it will be seen that there are the following additional commissures, the functions of which have been referred to at different times in the course of this article.

(iv.) From the visual word-centre to the glosso-kinæsthetic centre (*e e*).

(v.) From the auditory word-centre to the cheiro-kinæsthetic centre (*f f*).

(vi.) From the cheiro-kinæsthetic centre to the auditory word-centre (*f′ f*).

(vii.) From the cheiro-kinæsthetic centre to the visual word-centre (*d*).

(viii.) There is another important commissure, not represented in this diagram, by means of which the right auditory word-centre is enabled to exercise some amount of influence upon the left glosso-kinæsthetic centre in most persons, and a much larger amount of influence in some persons when the left auditory and visual word-centres are destroyed, as seems to be shown by the fact that some of these patients preserve a fair amount of speech, though mostly of a paraphasic type.

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The literature of this subject is enormous, but a very full bibliography, in chronological order, and extending over twenty pages, will be found in Mirallié's work, *De l'aphasie sensorielle*, Paris, 1896. In the writer's work, *A Treatise on Aphasia and other Speech Defects*, 1898, the whole subject is treated in a more complete manner than was possible in the present article, and is illustrated by details of over one hundred cases.

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IMPEDIMENTS OF SPEECH

IN this country the word "stammering" is used loosely for all f of speech defect, but especially as synonymous with stuttering. In literature, however, "stammering" (Stammeln) is never employed sense of stuttering (Stottern), but always to indicate other f [illegible] of impediment of speech. In order to avoid confusion, therefore, the word "stammering" will not be used in this article.

I. STUTTERING.—**Definition.**—A spasmodic affection of the muscles concerned in articulation, occasioned by erroneous nervous control, leading to a sudden check in the utterance of words, or to a rapid repetition of the literal sound in connection with which the difficulty arises.

History.—It is only during the last half-century that stuttering has been looked on as a complaint worthy of the attention of the physician. The first scientific treatment of the subject was contributed by Dr. Neil Arnott, in 1827, in an article on "Voice and Speech" in his *Elements of Physics.* In France, about the same time, Columbat recognised the nature of the complaint, and devised appropriate exercises to overcome it; almost simultaneously the first scientific account of the subject was published by Schulthess in Zurich. These papers, unfortunately, had but little influence, and the subsequent literature on the subject is full of the most grotesque notions of the pathology and treatment of the disorder. In 1840, under the influence of Dieffenbach, a brief period of surgical enthusiasm occurred, and extensive divisions of the lingual muscles were practised by many surgeons. The enthusiasm subsided in a few years owing to the number of fatal results, and this irrational procedure was entirely abandoned. Now, after many years, investigators are returning to the views and methods of Arnott, Columbat, and Schulthess, and elaborating them. The work of Kussmaul, Gutzmann, and Wyllie has placed the knowledge of the subject on a sound scientific basis.

Mechanism of normal articulation.—The mechanism of articulation requires particular consideration, as familiarity with it is absolutely necessary for the intelligent treatment of these disorders of speech. The production of articulate speech requires the orderly co-operation of three muscular mechanisms: (i.) the respiratory apparatus for supplying a blast of air; (ii.) the larynx for transforming the blast of air into voice; and (iii.) the musculature of the lips, tongue, and palate, for altering the shape of the passages so as to modify the voice and differentiate sounds into words. Not only must the component muscles of each of these delicate mechanisms act in perfect concert, but there must be absolute co-ordination of each of these mechanisms with one another. It is the difficulty of performing the necessary movements of the tongue and lips which usually obtrudes itself on a stutterer's attention, but, as we shall see, the essential difficulty consists in securing proper co-ordination between the vocal and articulatory mechanisms.

The English alphabet, being both defective and redundant, must be reconstructed on a phonetic basis, the various literal sounds being grouped according to the position in the mouth at which the mouth is modified to produce them. Several consonantal sounds (for example, *th*, *ph*, and *sh*) have to be added, and those which are really consonantal diphthongs, such as *q* (*kw*) and *j* (*dzh*), have to be omitted. The most convenient and scientific grouping of the consonants is that devised by Dr. John Wyllie of Edinburgh, which is here reproduced. The section on stuttering in his work on *Disorders of Speech* is by far the most thoughtful and suggestive consideration of the subject yet published.

Consonants

(From Wyllie's *Disorders of Speech*)

	Voiceless Oral Consonants.	Voiced Oral Consonants.	Voiced Nasal Resonants.
Labials. (1*st Stop Position.*)	*P* (W)[1]	*B* W	M
Labio-Dentals.	F	V	
Linguo-Dentals.	Th S	Th Z	
Anterior Linguo-Palatals. (2*nd Stop Position.*)	Sh *T* (L)[1]	Zh *D* L R	N
Posterior Linguo-Palatals. (3*rd Stop Position.*)	*K* H or Ch	*G* Y (R)[1]	Ng

The *vowel sounds* are grouped most conveniently according to their phonetic value rather than according to the letters; for in English each letter is pronounced in several different ways. The different sounds are best remembered by the mnemonic given by Pitman :—

Long.—Half pay she thought so poor
Corresponding Short.—That pen is not one foot.

I is omitted from this list, not being a simple vowel sound, but a diphthong and equivalent to *ah-ee*. *Y* as a vowel is closely allied to *i*, and *w* to *u*. It is a matter of dispute whether *h* should be regarded as a special consonant or as an unmodified expiration. Wyllie's view that it is a guttural allied to the Scotch *ch* is probably correct. It is an occasional cause of stuttering.

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[1] The voiceless *W* and the voiceless *L* have been given above within brackets, the former being now almost confined to Scotland, and the latter being peculiar to Wales. The burring or uvular *R* is also given within brackets.

For the intelligent direction of treatment it is essential that the medical attendant shall make himself perfectly familiar with the exact position of the lips and tongue requisite for the production of the various vowels and consonants. This he can best do by observing the movements and position of his own tongue, aided by the description in standard text-books of physiology.

The part played by the laryngeal and buccal musculatures respectively in producing the various sounds must not be overlooked. The vowels are mainly laryngeal sounds, modification of the sound for each vowel being effected by altering the shape and volume of the buccal cavity as a resonating chamber.

The consonants are mainly buccal sounds, but in many there is an important laryngeal element. It will be noticed that in Wyllie's physiological alphabet the consonantal sounds are grouped into "voiced" and "voiceless," a distinction of great importance in treatment. In the voiceless consonants—such as *p*, *t*, and *k*—there is no vocal element, but a nearly noiseless blast of air. In the case of the voiced consonants—such as *b*, *d*, *g*, and *m*—sound is produced in the larynx each time that the consonant is uttered. This can be easily realised by saying aloud *b-b-b-b*, without employing any vowel, when it will be found impossible to pronounce the consonant without using the voice, and the result may be contrasted with the almost silent production of the corresponding voiceless consonant *p-p-p-p*. This grouping corresponds to the division of the letters by the grammarians into "sharp" and "flat"; the voiceless group containing the sharp consonants, while the voiced group includes the flat consonants, the nasals, and of course all the vowels. The distinction between the voiced and voiceless consonants may also be demonstrated by the following plan recommended by Wyllie. Pronounce the word "wonder," in which all the consonants are voiced, in a low monotone. The vocal element produces a continuous murmur, which can be kept on indefinitely by repeating the word. If now the syllable "ful" containing the voiceless letter *f* be added, and the word "wonderful" be pronounced in a similar way, a break will at once occur in the vocalisation of the word at the letter *f*.

Causation.—Little is known with certainty of the causes of this disorder. Direct *hereditary transmission* does not appear to play a large part in the causation, but a neurotic family history is usually to be obtained, and, apart from the speech disturbance, the patient often betrays a neurotic tendency. *Sex* appears to have an important effect, the large majority of cases occurring in boys. *Imitation*, as from a stuttering nurse, is an occasional cause. *Adenoid vegetations* have been held responsible for the trouble, but although they are often present in stutterers, there is no evidence that they can directly give rise to it.

The affection is rarely congenital, but comes on either in early childhood or at some epoch in a child's life, such as going to school, the period of second dentition, or of puberty. A stutterer who has recovered may relapse under similar circumstances; such as going to a boarding school, or to live abroad, where he has to speak an unfamiliar language. Or it not infrequently begins during convalescence from some acute disease, particularly from diphtheria and measles.

Pathology.—The complaint must be regarded as a functional nervous disorder, without any structural changes, leading to erroneous action of the muscles concerned in speech. Although local conditions of the throat may increase the tendency to stutter, they are but contingent direct causes, as is clearly proved by the facility with which almost all stutterers can sing and intone.

In most cases the larynx is kept closed while the muscles are acting powerfully, the result being that the patient stands speechless and becomes very red in the face. In other cases the vocal cords are properly adjusted for the production of voice, but the expiratory muscles are not properly brought into play at the proper moment. In a third group vocalisation by the larynx occurs normally, but, owing to spasm of the lingual or labial muscles, articulation is checked. It is important to remember that in the majority of cases the laryngeal muscles are at fault.

Symptoms.—The most characteristic symptom in stuttering is an intermittent inability to emit certain sounds, the disability being noticed only when the patient speaks in a conversational voice, and being rarely present when he sings or intones. The difficulty in articulating is mainly experienced when the difficult consonant occurs as an initial letter, no hesitation being noticed when the same letter occurs in the middle of a word. The actual phenomena observed vary greatly in different individuals. Most frequently the patient has a habit of speaking in a monotonous and rapid fashion, with his chest almost empty. As he is speaking, the flow of words is suddenly interrupted, and he hesitates, with every appearance of violent effort, before pronouncing the difficult consonant. Suddenly the stumbling-block is removed, and the word is pronounced with an explosive jerk, and the subsequent words tumble out rapidly, in a succession of jerks, "like water being poured out of a bottle," until checked by a fresh stutter. This jerky mode of speech often persists in adult life long after all traces of the original stutter have disappeared. The impediment is notoriously worse when the stutterer is nervous or hurried, as when getting a railway ticket; it is also worse when he is in bad health. It is a characteristic of stutterers that they will always persist in their efforts to pronounce the difficult letter, and will never substitute a more easily pronounced synonym, even if it be suggested by a bystander.

Stuttering is very rare with vowels, and is most common with the explosive consonants, *p*, *b*, *t*, *d*, *g*, *k*. Not infrequently the stuttering appears to be induced by nervousness because of a tendency to lisp on some letter which follows the initial letter. Several patients always *stuttered on the initial letter if the word subsequently contained an r, which they pronounced as w; and the stutter quite disappeared when, by diligent practice, they had learned to pronounce the r with facility.* A clergyman became incapacitated for work because he stuttered at the beginning of any word, such as *behold*, which contained an *h*, owing to nervousness lest he should omit the aspirate.

In the majority of cases the patient remains silent during his attempt to speak; but occasionally he manages to pronounce the letter, and continues to repeat it, speaking, for instance, of a *b-b-b-ath*, *b-b-b-un*. This, however, is more common in anecdote than in real life. Occasionally the impediment is aggravated by the occurrence of *associated sounds* with the stutter, the patient emitting unpleasant little whoops, grunts, or whimpering sounds during his efforts to speak. Not infrequently these noises are "tricks" that were used, in the first instance, to correct a stutter, and then became grafted upon it. A patient finds, for instance, that by drawing his breath and starting again he can avoid stuttering on the difficult consonant; but in doing this there is great danger of vocalising with the short inspiration, and giving rise to a little whoop or crow (the "drawback phonation" of Wyllie), which is interpolated in his conversation, and may persist long after the stutter has been conquered. In some published cases a meaningless syllable or word, such as "hedera," "nana," etc., is interpolated in the same way, and is very difficult to get rid of.

In other cases there are *associated movements* which are even more trying to bystanders than the sounds, but are fortunately infrequent. They are usually confined to facial contortions, or to a sudden jerk of the head; but occasionally there are violent gestures of the arms. In one case the whole body became rigid, and was slowly rotated, with the face upturned, the spasm suddenly ceasing when the word was ejaculated. The movements in this case, and in others which have been reported, bore a superficial resemblance to the beginning of an epileptic fit, and confusion is specially apt to arise when the face and neck of the stutterer become congested during his violent expiratory attempts.

Method of investigation of a case.—The chest of the patient should be uncovered, and he should be made to read aloud from an unfamiliar prose book. As he reads, any faults of the respiratory movements should be noted, and any associated movements of the face or limbs observed. Attention should be closely given to the character of the stutter, and a mark should be made under each word at which he hesitates. It will be found that, as a rule, a few initial letters only give trouble, the rest being articulated without effort. The difficult words should be compared, to see if they have some common character, such as the inclusion of some letter which the patient habitually pronounces with a lisp (*vide supra*).

Prognosis.—In most of the cases there is a tendency to spontaneous cure. The prognosis is worst in persons of a nervous and sensitive temperament. Recovery is hastened in all cases by systematic intelligent exercise.

Treatment.—There is no royal road to recovery for stutterers. Although the plan of treatment has to be varied for each individual case, the common feature of all is intelligent, regular, long-continued practice. The various mechanical contrivances which have been devised are seldom of the slightest value. These have been largely inspired by the tradition that Demosthenes was a stutterer, and cured himself by shouting with pebbles in his mouth. He certainly had some impediment in his speech, but most probably it was rather of the nature of a lisp. The other means adopted by Demosthenes, however, are better worthy of recall. He retired from society until by patient practice he should have conquered the defect, and he shaved half of his head in order to prevent his return to his wonted haunts until he had succeeded. Surgical operations on the organs of speech have proved useless, and occasionally disastrous, as might have been expected.

For checking the stutter daily reading exercises are essential; and at first, at any rate, should be supervised by the medical attendant. Where this is impossible, a relative, who has been instructed in the principles of treatment, should always be present to see that the directions are carried out.

(i.) The chest must be kept well filled with air—a very difficult habit for these patients to acquire.

(ii.) The patient must speak slowly, with a full resonant voice, enunciating all the consonants distinctly. He must avoid the sing-song style into which stutterers often fall, and must try to acquire a deliberate rhythm in ordinary conversation.

(iii.) When he comes to a word on which he tends to stutter, he must remember that in order to check it he must direct his energies, not to articulation, as he instinctively tries to, but to vocalisation. He should try to raise his voice as if he wanted it to carry farther. If he stutter on one of the "voiced" consonants such as *b*, he must produce the vocal element of the letter. If the consonant is "voiceless," as *p* in *pat*, he must attempt to vocalise the *at*, and he will find little difficulty in prefixing the *p* as the *at* is uttered.

Gymnastic exercises are valuable to correct the imperfect filling of the chest, which is so common; and singing exercises are particularly useful, they give confidence to the patient, and teach him how to vocalise more effectually. Associated movements are best checked by making the patient practise his reading exercises before a mirror, so that he may be aware of them himself, and endeavour to suppress them.

But to obtain very definite improvement considerable time is required. Even after a month of diligent practice the patient may have made little advance, and be inclined to relinquish the struggle. But, as with learning to swim, when once confidence is restored the rest quickly follows. It is therefore a matter of great importance that a boy should be encouraged, and protected from the scoffs of his fellows while under treatment. It is often of great advantage if he can be taken into the house of a medical man for some weeks.

The general health may require attention, but no drugs have any effect in curing this troublesome complaint.

II. LISPING.—A defect in articulation caused by the indistinct enunciation of certain consonants, or by the substitution of consonants other than the proper one. These defects may be due to deformities in the conformation of the mouth, or to clumsiness in the use of the tongue and lips. It is seen as a normal condition in infants who are learning to speak, but as a rule the difficulty is soon overcome. The most common substitutions in young children are *t* for *k*, *th* for *s*, *f* for *th*, and *r* for *w*. In a few persons the defect persists to adult life and forms a personal characteristic. The most common defects in adults are inability to roll the *r* sound, with a tendency to substitute *w*, and the substitution of *sh* or *th* for *s*. Lisping is often acquired by affected adults, and paraded as evidence of superior breeding. Some years ago *w* was substituted in this way for *r*, and at the present day many persons affect an inability to pronounce the final *ng*, and substitute *n*.

The difficulties met with in infancy are again encountered in learning a foreign language. For a long time certain consonants present very great difficulty, and may never be spoken with facility. The difficulty of the ordinary Frenchman with the English *th*, and of the Englishman with the German gutturals, are cases in point. Not infrequently the difficulty is insuperable, and the peculiarity of pronunciation becomes a national characteristic. During the "Bread and Cheese Riots" in London, in the reign of Richard II., the mob attempted to exterminate the immigrant traders and skilled workmen from the Hanse Towns, by surrounding the streets in which they lived, and putting to death all who pronounced the words "bread and cheese" with an accent. The use of the test word "Shibboleth" during a struggle between some Jewish tribes affords another example.

Diligent practice alone can overcome the difficulties in the pronunciation of the consonants in our own or in a foreign language; but this may be facilitated by a knowledge of the correct positions of the tongue or lips. The teacher must be familiar with them himself, must show them to the patient, and then get him to imitate him.

III. IDIOGLOSSIA.—This name was given by the late Dr. Hadden to a very severe form of lisping, in which the patient's speech is so altered that he seems to be speaking in a language of his own. On carefully analysing the words, however, it is found that many of the letters are not pronounced by the patient, who substitutes for them others, most usually *t*, *d*, or *n*.

A good illustration is the example of a boy under my own care who thus repeated the Lord's Prayer:—"Ouë Tahde na ah in edde, anno de Di na, I tidde tah, I du de di on eet a te e edde, te ut te da ouë dade ded, e didde ouë tetedde a ne ahdin to te tetedde adase ut, ne no te tetate, ninne utte enu, to I ah te nini, pouë e dordy, to edde e eddé, Amé." Strange as this looks and sounds, it will be found that there is method in it. The patient was unable to pronounce many of the consonants, and substituted for them *t*, *d*, or *n*.

The condition cannot be very rare, as fifteen cases have come under my own notice. As with stuttering, about two-thirds of the patients are males. They are usually bright and quick, and, as they have great facility in communicating their ideas and wishes by facial expression and pantomime, they take little pains themselves to overcome the defect. The peculiarity of speech is usually present from the time when the child begins to speak, and it tends to disappear after the age of eight, if the child realises the inconvenience of the defect, and takes pains to overcome it. In all the cases seen by myself there was a well-marked neurotic family history. One patient had had an attack of left hemiplegia in infancy, from which he had completely recovered; another was suffering from valvular disease of the heart. None, except the first mentioned, showed any indications of organic disease of the brain.

The auditory word-centre appears to be perfectly normal, since the children learn quickly to understand what is said. There seems to be some imperfection in the training of the motor centre in the frontal lobe, which, as Dr. Bastian has shown, is trained through the medium of the auditory centre; but of the exact nature of the irregularity we are at present ignorant.

On carefully testing the patients with the sounds given in the physiological alphabet (p. 449), the exact sounds which are defective and those which are substituted for them will be demonstrated. The posterior linguo-palatals *k*, *g*, and next to them *f*, *v*, and *r*, are most frequently affected. The labials and linguo-dentals nearly always escape. The average number of consonants which twelve patients failed to pronounce was eight; and several others, which could be pronounced as initials, presented an insuperable difficulty when they occurred as final letters. In rapid automatic speaking they almost all substituted *t* or *d*; the former for voiceless, and the latter for voiced consonants: when speaking slowly and with care, the substituted sounds are more varied. The vowels have not been affected in any of the recorded cases.

Treatment.—It must be remembered that when once the child has become accustomed to express his thoughts by these erroneous sounds, it is as difficult for him to express them in our way as to acquire a foreign language. And just as it is important on going abroad to learn a foreign tongue to keep from the society of one's fellow-countrymen, and to avoid speaking English, so it is most desirable that the patient shall be isolated from those who have learned to translate his jargon. He should be sent away from home if possible, and his training undertaken by a relative or some skilled teacher—preferably some one accustomed to instruct deaf mutes in speaking; or he may be sent to a school for deaf mutes. The teacher must show him the exact position of the tongue in the pronunciation of the various consonant sounds, and get him to imitate it. At first only the consonant sounds themselves should be practised, then syllables; but the patient should not try to pronounce words and sentences until considerable facility in the enunciation of the simple sounds has been acquired. In severe cases this takes several months; in slighter cases much less. The progress depends largely on the earnestness with which the patient enters into the exercises, and also, of course, on the patience and capacity of the teacher; but the ultimate results are usually most satisfactory.

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N.B.—A full bibliography will be found appended to Gutzmann's *Storungen der Sprache.*

W. S. C.

CONCUSSION OF THE BRAIN

Although the more severe cases of concussion of the brain come immediately under surgical treatment, a large number of the slighter cases, escaping treatment altogether at first, in the course of a few days after the accident fall into the hands of the physician in consequence of symptoms which are set down by the patient to causes unconnected with the injury, to which, however, they are really due. Indeed, my own experience tends to show that medical men are not always alive either to the importance of these symptoms, or to the relation which they bear to an injury which in some instances may have appeared to be trivial. This want of vigilance appears to be due mainly to the persistence of the teaching of most of the surgical text-books, which insists upon pronounced loss of consciousness as a necessary symptom of concussion of the brain. Without ignoring such points as may be usefully discussed in relation to concussion of the brain generally, it is my intention to deal more especially with the minor cases. For this no apology is offered, since it is certain that the apparently trivial nature of some of these cases leads finally, for reasons which will become manifest, to their proving of more importance than many of those cases which are attended immediately upon the occurrence of the injury by much graver symptoms, as, for example, by prolonged unconsciousness.

The consideration of the subject must of necessity be from a purely clinical standpoint, seeing that in the conditions dealt with in the present article opportunities for pathological investigation are rarely if ever forthcoming.

For practical purposes cases of concussion of the brain may be classified as follows :—

1. Those in which unconsciousness, following immediately upon injury, lasts for a period of one hour and upwards.
2. Those in which unconsciousness persists for periods varying from a few minutes to an hour.
3. Those in which the loss of consciousness occupies a few seconds only.
4. Those in which no loss of consciousness occurs at all.

Classes 1 and 2 represent the severe forms of concussion as described in the various text-books, and, by reason of their grave and unmistakable symptoms, are sure of immediate treatment. Classes 3 and 4 are the milder forms which, in consequence of the slightness of the symptoms, frequently escape treatment altogether in the first instance; especially as unconsciousness is very transient or entirely absent. Hence cases of this class, merely from primary neglect on the part of the patient, or from failure on the part of the practitioner to appreciate the possible importance of the condition, may in the end have far graver results than the severe cases in which treatment as a matter of course is rigidly enforced from the beginning.

Loss of consciousness, as such, is no necessary concomitant of concussion of the brain. Injury, if sufficiently severe, will cause complete unconsciousness for periods varying from minutes to hours or days. Many cases, however, occur, that with no less accuracy may be described as examples of concussion, in which, the injury being slight, the loss of consciousness may either be so transient as to escape notice altogether, or may not occur at all—a feeling of being "dazed" or confused for a few moments being all that the patient notices, excepting perhaps a temporary sensation of giddiness.

In these milder cases the symptoms are at times mistaken for indications of "biliousness," or of other ailments with which they are not really connected; their true cause being ignored or overlooked. Such cases are therefore of much interest, and, considering the serious nature of their occasional issue, have not received the general attention they merit.

The degree of loss of consciousness or brain confusion in concussion depends mainly upon the severity of the injury; and in severe cases the whole cranial contents are of necessity more or less uniformly shaken and jarred. In the slighter cases, however, not only is the loss of consciousness merely partial or altogether wanting, but, as it appears from my own experience, the concussion need not necessarily affect the whole of the intracranial nervous apparatus, but may be limited either to the cerebrum proper, or to the medulla and pons with their connections lying in immediate relation to the base of the skull, especially to the basilar process. It also appears that the symptoms arising in concussion of these two portions of the brain in cases of slight injury differ sufficiently to justify their separate consideration under the heads of concussion (*a*) of the brain proper (cerebrum), and (*b*) of the parts extending from the mesencephalon above to the cerebro-spinal junction below.

The situation of the intracranial damage seems to be determined by the kind of injury received. Direct blows on the skull—as, for instance, from a hammer, or a projecting branch of a tree—are followed by concussion of the brain proper; whilst indirect injury, such as may occur in falls on the buttocks—the trunk being at the same time vertical or nearly so—may be followed only by damage to the parts immediately above the cerebro-spinal junction, the latter kind of damage being consequent on the jamming of the parts against the bony surface, with which they lie in contact, by the sudden impact of the heavy structures above, at the moment when the body is arrested in its fall. I am aware that exception may be taken to the possibility of differential concussion of portions of the intracranial contents in the manner suggested; but it is, I believe, a fact, which admits of no denial, that the two kinds of injury (direct and indirect) produce symptoms which are so far distinct from each other as to be easily recognisable if a little care be used in their investigation.

It will be found further, in many instances of severe concussion with profound unconsciousness, that the symptoms after the reactionary stage will by degrees conform to the type characteristic of damage by direct or indirect injury, as the case may be. The appreciation of the difference in the symptoms of these two conditions is of small importance in relation to treatment, since all cases of concussion must be managed upon the same principles; but in the matter of prognosis it is of considerable moment, as will presently be shown.

CONCUSSION FROM DIRECT BLOWS ON THE CRANIUM—(Concussion of Cerebrum).—After a direct injury to the skull, if the violence be sufficiently great, the ordinary symptoms of concussion with profound unconsciousness must follow immediately. In the slighter cases, when the loss of consciousness is very transitory or altogether wanting, no symptom may follow directly upon the injury sufficient to attract the patient's attention, or at all events to lead him to seek treatment at the hands of a medical man. Sooner or later, however, after the lapse of a few hours or perhaps of days, symptoms almost always arise which lead to the summoning of advice, although they may not then have any connection in the mind of the patient with the apparently trivial accident met with a short time before.

Symptoms.—The first symptom to attract attention is usually pain, with or without giddiness, nearly always associated with restlessness and constipation; a combination which leads the patient to suppose that the trouble is due to "biliousness." Careful examination at this stage elicits the following symptoms:—Pain, giddiness, a general feeling of sensitiveness about the cranium, an unnatural aspect of face, a distinctive character of pulse and a peculiar mental mood. The pain is more or less acute, extending over the vertex and across the brow; it is increased by movement, and is worse after taking solid food. The giddiness bears a direct ratio to the amount of pain, and is influenced by the same conditions. The peculiar facial expression, although not always sufficiently well marked to attract attention upon superficial observation, is nearly invariably present; it is difficult to describe it better than by saying that the patient has a dazed and sometimes half-frightened look. The pupils as a rule are irritable; but they may be sluggish, and then are often unequal: photophobia, in a minor degree, is not infrequent. The symptom which never fails in constancy and quality is a peculiar character of the pulse which is common to every case of concussion of the brain, severe or slight; and is never absent, excepting temporarily during the reactionary stage, in very severe cases with profound unconsciousness. This pulse, when felt by the finger in the ordinary way, seems fairly good, and at first perhaps suggests nothing worthy of note; it comes up quickly under the finger and seems sufficiently strong; but the slightest pressure reduces it to nothing; it is extraordinarily compressible—the phrase "action without power" fairly describing its character. In cases of the class under discussion this peculiarity of the pulse may be the first symptom to attract attention, and the last to disappear; the rate varies from 70 to 100 per minute, and the tendency is to rapidity rather than the reverse. The skin is warm to the touch and inclines to dryness; very rarely is there any unnatural moisture. Intra-ocular changes (choked disc) occasionally occur, but are temporary: the reflexes (knee-jerk, etc.) are generally unaffected.

Wasting of the muscles occurs, especially of those of the extremities, and is much more manifest if the patient is kept to bed; but it does not, as a rule, exceed in degree that which may naturally be produced by disuse.

The mental mood inclines to irritability and antagonism; the patient resents the restrictions necessary for his proper treatment, and tends to become over-critical, and sometimes suspicious of those about him.

CONCUSSION FROM INDIRECT INJURY—(Concussion of the parts immediately above the cerebro-spinal junction).—Indirect injury, if sufficiently violent, as in the case of direct blows, will cause general concussion with profound unconsciousness, varying in degree with the extent of the damage.

In the slighter cases the immediate result of the injury is frequently no more than a very temporary mental confusion without loss of consciousness. These apparently trivial cases are again, as in the milder forms of those resulting from direct injury, of great importance, as the insignificance of the initial symptoms sometimes leads to neglect.

After the passing away of the mental confusion at the moment of the injury the patient so far recovers that he is not usually led to seek advice until some hours, days, possibly a week or even a fortnight later; when pain and feelings of general depression call attention to an unsatisfactory condition of health, which is commonly attributed to gout by the patient himself, and sometimes even by the medical practitioner.

Symptoms.—The main symptoms elicited at this stage are—pain, a general feeling of heaviness about the head, restlessness at night, sweating after slight exertion, the peculiar pulse, evidence of rapid muscle wasting, and a peculiar mental mood.

The pain is dull and limited to the parts about the occiput and nape of the neck; very rarely does it extend above the occipital region: with the pain and limited to the same area is a feeling of heat, which is often so much in excess of the pain that it is the only discomfort referred to. The head is held somewhat stiffly, and is as a rule bent a little forwards. The parts below the occipital protuberance and about the nape of the neck are frequently too sensitive to the touch. The expression, beyond suggesting fatigue, is natural. Photophobia is rarely present, but muscæ are often complained of, and "singing" in the ears frequently causes annoyance.

The feeling of heaviness is general and amounts at times to an uncontrollable lethargy. With this is a peculiar mood of mental apathy; the patient feels ill, but does not resent his illness, and shows no great desire to escape from it; subsequently there is a strong inclination to lapse into contented invalidism. The pulse is of the character already described, and inclines to infrequency, sometimes slowing down to 30 beats to the minute. In a cabman recently under my care in St. George's Hospital the pulse-beat fell to 28 in the minute. In some cases this slowness of pulse becomes practically permanent. The temperature remains nearly normal, as a rule; if any change occur, it is usually to fall below the normal. The urine sometimes contains sugar. The sweatings are fairly constant and characteristic; they follow any, even slight, exertion, and are always ushered in by the feeling of heat about the back of the head already referred to; at night they occur spontaneously and are profuse, coming on generally as the patient awakes from sleep—especially if a change from the lying-down to the sitting-up position is rapidly made. General muscular wasting is remarkable, and sometimes so rapid as to become very marked in a few days; it occurs whether the patient be confined to bed or not, and in the former case is altogether in excess of that which may come of mere disuse. As the appetite is usually good, and sometimes voracious, adiposity often rapidly follows the muscular wasting.

The reflexes (knee-jerk, etc.) are not affected, as a rule; the knee-jerk, however, sometimes inclines to slight exaggeration, and in rare cases may be very excessive.

Comparison of the symptoms described above, as resulting from direct and indirect injury of the milder sort, indicates distinct differences which can hardly be mistaken in exemplary cases. It is clear, however, considering the great difference in the degree of violence and in the conditions under which it is inflicted in different cases, that in many instances (indeed, in the majority) the symptoms must at first so much overlap one another as to lose their distinctive characteristics; although, as has already been stated, they tend strongly, as the condition of the patient improves, to conform to one type or the other. The importance of this fact in prognosis is considerable. The resemblance of the symptoms, in the milder class of cases, to certain forms of illness from liver disturbance, gout, and the like, requires no analysis here. It may, however, be excusable to suggest the wisdom of paying more attention to the possibility of some slight injury of the head as the cause of illness associated with vague, ill-defined pain and discomfort about that part, rather than to assume at once that the symptoms have some gouty or dyspeptic origin. I have less hesitation in suggesting this somewhat obvious precaution, as cases of the kind have come under my personal observation in which symptoms clearly due to an injury, too trivial to attract the patient's immediate attention, were considerably aggravated by the treatment for gout—including in one case a visit to Aix-les-Bains and other European "watering-places"—which could hardly have been advised had the recent injury been known to those concerned in treating the patients. The association of the peculiarity of pulse described, with a history of injury to the head, however slight it may appear to have been, should leave no room for a misapprehension of the nature of the case.

Prognosis.—The prognosis, with regard to the life of the patient, in severe concussion of the brain depends, in the first place, upon the gravity of the injury and the degree of shock; and, secondly, upon the efficacy, or the reverse, of the treatment carried out. Comparatively few cases of concussion alone prove immediately fatal (from shock); but gross organic brain lesions, such as laceration or hæmorrhage, may cause death, either immediately or very shortly after the accident. If a rapid rise in temperature and pulse-rate occur, danger to life may also arise in severe cases during the stage of reaction, the cause of death in these circumstances being generally acute meningitis; but acute meningitis is rare in concussion without fracture of the skull, or some gross lesion of the brain or membranes. In young and delicate subjects tuberculous meningitis may supervene, and cause death in cases in which the temperature has been high in the reactionary stage; this result, however, rarely happens unless the tuberculous tendency is strong.

In simple concussion the gravity of the case increases step by step with the period during which profound unconsciousness lasts; and it is undeniable that recovery from profound insensibility is more rapid, and the stage of reaction less severe, in cases complicated with scalp wounds which give rise to free bleeding; a fact which has an important bearing upon treatment. The vomiting which ushers in the stage of reaction is usually salutary, but in very severe concussion in feeble subjects it may be followed by syncope and fatal collapse. The progress after the stage of reaction depends almost entirely upon the efficacy of the treatment. However severe the case, complete recovery may be reasonably anticipated under proper management; provided, of course, that no gross organic lesion exist. On the other hand, if the treatment be inadequately carried out, the recovery will be partial; the defect in recovery depending upon the want of proportion between the treatment and the gravity of the case. A very severe case treated adequately will be more likely to end in complete recovery than a comparatively mild one imperfectly managed; the commonest cause of imperfect recovery being the return of the patient too soon to the ordinary habits of life.

In the milder cases no immediate danger to life arises, but it is common for the recovery to be only partial, and this on two accounts:—(i.) the neglect of all treatment at first; and (ii.) the failure to continue the proper restrictions long enough. The first of these causes is the more important, because treatment is sometimes neglected until the patient is beyond the stage in which complete recovery in the full sense is possible. Complete recovery means the return of the patient to the normal condition of health in all respects; partial recovery includes every degree, ranging from helpless chronic invalidism, or mental derangement, to a state so near the normal condition of health that the only symptom detectable may be the persistence of the peculiarity of pulse described earlier in this article. Slight cases of concussion, if treated properly at once, invariably end in complete recovery; but neglected cases of this class afford the majority of imperfect cures. A certain number of slight cases do undoubtedly end in recovery without any treatment at all, but this number is very small indeed; in nearly all the apparent recoveries after neglect of treatment some trace of the damage shows itself sooner or later. The patient is rarely quite restored to the same condition as before the accident; age advances prematurely; energy is to some extent lost; tastes change, and peculiarities, sometimes of an objectionable kind, appear which are quite foreign to the original disposition of the individual.

Speaking generally, it may be considered that in cases in which irritability and antagonism are amongst the early symptoms (that is, in the type of case described under the head of concussion from direct injury), the results of imperfect recovery tend to the manifestation of vicious peculiarities, such as ill temper, criminal inclinations, insanity, and proneness to suicide. On the other hand, in cases like those described under the head of concussion from indirect injury, in the earlier stages of which all symptoms of resentment or annoyance are conspicuous by their absence, the patients are prone, when recovery is incomplete, to sink into contented invalidism with a melancholy tendency, to become gray early and prematurely aged, or to assume entire indifference to calls of business or duty. These patients display no vicious inclination, nor any sign of lunacy or suicidal tendency; but they seem incapable of applying their minds to any useful end. A curious form of partial recovery, which may occur in any case of concussion, is that in which, while the health is generally good, the patient completely changes in disposition, sometimes in a most striking manner. It is not unlikely that amongst certain classes neglected cases of slight concussion are more responsible for indifferent health, lassitude, and apparent indolence than is commonly suspected. The occurrence of temporary glycosuria in some of these cases is a symptom which does not appear to affect prognosis; it would, however, be a matter of great interest to determine whether injuries of the brain, in which glycosuria follows immediately, dispose to the occurrence of diabetes at a later date.

Treatment.—The treatment of all cases of concussion of the brain, whether they be severe or slight, may conveniently be dealt with in three stages: (i.) immediately after the injury; (ii.) during the period of reaction; (iii.) during the period subsequent to the reaction stage.

In a general sense, it may be held that incompleteness of recovery in slight cases depends for the most part upon inadequate treatment immediately after the injury; and, in severe cases, upon imperfect management during the period subsequent to the reaction stage.

(i.) *Treatment immediately upon the receipt of injury.*—Absolute rest in bed is the first essential indication; and a mercurial purge should be given at once, 5 grains of calomel (in adults) sprinkled on the tongue being the most effectual, and, indeed, in profound unconsciousness, almost the only practicable means of attaining the desired result. The nourishment should be sustaining and of a fluid kind; alcohol is inadmissible unless indicated by special symptoms. In severe cases no difficulty, of course, arises in the treatment of the early stage; but in the slight cases, where the loss of consciousness or mental confusion is very transient, leaving the patient for the time being apparently well, great and sometimes insuperable difficulty is met with in inducing the injured person to submit to the restraint and inconvenience which adequate rest entails. Nevertheless, it is impossible to over-accentuate the importance of immediate rest, even in the slightest cases; for it is upon this immediate quiet that the ultimate issue of the case almost always depends. Although sometimes it may be impossible to induce the patient to pursue the proper course, the practitioner has at all events no further responsibility if his advice is ignored, or followed imperfectly.

Obvious as the indication may appear, I make no apology for urgently insisting upon the necessity in these mild cases for complete rest at once, or as soon as possible; from my own experience it is clear that there is a conviction amongst not a few practitioners, that in such apparently trivial cases nothing is necessary beyond free purgation, a low diet for a few days, and a temporary abstention from alcohol, with perhaps some very short and incomplete rest—a line of treatment which directly favours incomplete recovery in its various degrees.

(ii.) *Treatment during the period of reaction.*—In severe cases the classical means, as described in the text-books, are still the main indications; that is to say, a continuance of complete rest, a low diet, and gentle purgation. A comprehensive discussion of the treatment of the complications which may arise at this stage—such as vomiting, rise of temperature, increase of pulse-rate, headache, and sleeplessness—is beyond the scope of the present article. The following points, however, are worthy of special attention: there is, as I have already said, no doubt that the severity of the symptoms in the reaction stage is, other things being equal, less in cases in which bleeding occurs from wounds received at the time of injury. It is equally certain from my experience that all symptoms during the period of reaction are diminished by a moderate blood-letting (say ℥vj. or ℥viij.) at its onset; this operation may safely and with advantage be practised in all cases in which there is distinct increase in pulse-rate or blood-pressure with or without rise of temperature. Indeed, I have good reason for believing from practical experience that, even in cases in which the symptoms of reaction are less marked, a small abstraction of blood by venesection is advantageous. One thing at least is certain, that nothing is so effectual in removing the headache, which is often met with at this stage, as the application of two or three leeches behind the ear, or to the nape of the neck.

It is interesting to note the difference in the estimation, by different surgeons and at various hospitals, of the value of the application of extreme cold by means of the ice-bag, or Leiter's tubes, in the treatment of concussion of the brain. Some years ago at St. George's Hospital the application of the ice-bag was a routine treatment in concussion cases, the head having been previously shaved. At the present time this treatment is altogether exceptional, in fact it has been discontinued. So far as my observation goes, the effect of cold is negative; for in a careful comparison of cases treated by (*a*) the ice-bag or Leiter's tubes, (*b*) by hot applications, and (*c*) without any local application to the head, it was found that the progress in parallel cases was step by step the same; and that if any material relief to pain or discomfort were afforded it was by the application of heat rather than of cold. There appears to be no objection to the discreet use of any sedative or narcotic, excepting morphine, which, unless called for by special circumstances, should be avoided.

Upon the treatment of the stage of reaction in the slight cases it is unnecessary to dwell, since the period of reaction, although it occurs in all such cases, is in them so slight or transitory as to be hardly noticeable. In fact, the treatment of all the slight cases may be said, for practical purposes, to begin with the stage subsequent to reaction; especially as these cases are rarely seen by the practitioner until the period of reaction has passed.

(iii.) *Treatment during the period subsequent to the stage of reaction.*—Upon the termination of the stage of reaction the complete rest, and modified diet (beef-tea, soup, milk, and the like) must be continued for a time, which necessarily varies in different cases. Upon the discretion of the practitioner in the management of diet, and in the selection of the proper time for discontinuing complete rest, the recovery of the patient for the most part depends. In this connection the principal points requiring consideration are the following:—

A. The time for the return to solid food.

B. The time for discontinuance of complete recumbent rest, and the time when the patient may be allowed to leave the bed and by degrees resume the habits of ordinary life.

A. *The return to solid food.*—Solid food should not be allowed until convalescence has fairly set in; and, in determining the time for making the change from a liquid to a solid diet, the condition of the pulse and the facial expression are the sole trustworthy guides. As soon as the dazed, vacant, or frightened look has left the face, soft solid food, such as boiled sole or whiting, may be given in small quantity; the recumbent position being strictly maintained, and the effect carefully watched. If, after food of this kind has been taken for three or four days, or in bad cases for a week or ten days, no ill effects follow in the form of dyspepsia, giddiness, headache, increase of pulse-rate, or rise of temperature, the same sort of food should be continued, the patient whilst eating being allowed to sit upright in bed. Should none of the ill effects mentioned then appear, white meat, such as chicken, may be allowed and continued for some days, when, if still no adverse symptoms arise, ordinary diet may by degrees be resumed.

If in altering the kind of diet in the manner indicated any of the symptoms mentioned supervene, the former diet should be resumed for a time, which must be determined to some extent by the severity of the unfavourable symptoms recurrent.

The period which intervenes before solid food can be taken with comfort necessarily varies according to (a) the severity of the case, and (β) the degree of the efficiency of the treatment adopted immediately after the injury; a period which may vary from a few days to months. In all cases it is necessary, upon the resumption of ordinary diet, to insist upon moderation; as it is a singular fact that after some of these injuries a most voracious appetite is awakened, which, if allowed to be indulged unchecked, may lead to untoward complications.

B. *The time at which the patient may be safely allowed to leave the bed, and by degrees resume the ordinary habit of life.*—As soon as solid food can be taken without giving rise to any symptom of discomfort, or without producing any change in the pulse or temperature, permission may be given for the patient to leave the bed for a short time daily. In allowing this freedom, it must not be forgotten that it is, as a rule, most difficult to induce a patient to return to recumbent rest; it is therefore important that this permission should not be given too soon. Speaking generally, no patient should be allowed to leave the bed until the pulse has resumed its normal character. The progress toward complete liberty of action on the patient's part must of course be gradual; the occurrence of vertigo, and especially of sweating, are certain indications that too much is being done, and whether they occur singly or together, should always be regarded as important warnings. If slight exercise is followed by no abnormal perspiration or other easily appreciable discomfort, it is generally safe to conclude, if the pulse is normal, that recovery will follow in the ordinary course.

In certain cases, which at their onset have been neglected, it will be found that the pulse permanently assumes the morbid characters described, and tends to become intermittent; in such cases, although an attempt to resume the ordinary habits of life must sooner or later be made, complete recovery rarely follows, the incompleteness of recovery varying from slight mental inertia, or slight loss of memory, to unmistakable dementia. The discussion of the various remote phases of partial recovery are beyond the limit of this article, and in substance will be found dealt with elsewhere.

WILLIAM H. BENNETT.

TUBERCULOUS MENINGITIS[1]

Definition.—Tuberculous meningitis is an acute disease depending on the invasion of the cerebral pia mater by the tubercle bacillus. The results of this invasion are the following morbid changes (which vary much in relative proportion):—the formation of miliary granulations along the sheaths of the vessels of the pia mater, the deposition of inflammatory lymph in the subarachnoid space, softening of the brain, acute hydrocephalus, fluid exudation into the spinal theca, and the formation of miliary granulations in the spinal membrane. These lesions of the nervous system are likewise accompanied by tuberculous deposit in one or more of the other organs of the body; some of this deposit may be contemporaneous with the lesions of the nervous system, but some of it is antecedent thereto.

History of the disease.—Our knowledge of the different anatomical characters of the disease, as set forth in the above definition, has been slowly won by successive workers. Robert Whytt (1768), in his observations on the "Dropsy in the Brain," although he had not recognised the specific nature of the affection, gave an excellent clinical picture of tuberculous meningitis; he insisted on its prodroma, and divided the illness into the three stages which, ever since his time, have been accepted. He first pointed out the important change in the pulse from quickness and regularity in the first stage to slowness and irregularity in the second; and that in the third stage the pulse rose to "feverish quickness and once more became regular." The anatomical feature which arrested his chief attention was the acute hydrocephalus, and by this title tuberculous meningitis was long known.

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[1] In preparing this article I have received much help from my friend Dr. Still, Medical Registrar of Hospital for Sick Children, Great Ormond Street.

In 1825 Senn made the important addition that the primary inflammation is at the base of the brain, and that the pia mater is the membrane invaded. The tuberculous nature of the disease was discovered by Papavoine in 1830. He described the tubercles as "plaques" and granulations, and indicated their distribution in the meninges, and their identity and association with tubercles in other parts of the body. Papavoine also made the important observation that granulations might exist in the pia mater without giving rise to obvious inflammatory reaction.

Barthez and Rilliet advanced the knowledge of the disease enormously, especially on the tuberculous side. They asserted that the prodroma are the expression of the tuberculous diathesis; they regarded the existence of tubercle in organs other than the brain as the most essential antecedent condition; they even went so far as to claim as tuberculous meningitis cases of fatal brain affection in children in whom no tubercle was detectable in the brain, but in whom tuberculous deposits existed elsewhere; and in the typical manifestation of the disease they insisted on the multiple pathology which has been indicated in the above definition.

By degrees the partial convulsions of the second stage of the disease were referred to cortical disturbance consecutive to the meningeal inflammation. Also the distinction was drawn, by Rendu and others, between the temporary paralyses, the pathology of which is not explained, and the permanent paralyses of which many examples have been recorded, and which appear to depend on more or less circumscribed softening.

It was also maintained by Hayem that, in addition to the softening from necrobiosis after vascular obstruction, there is a true diffuse, inflammatory encephalitis.

There has been much discussion on the nature of the hydrocephalus and the part played by it in the production of the pupillary changes, the paralysis and the coma. Some, at least, of the pressure symptoms, which originally were referred exclusively to the ventricular effusion, are now attributed to a general encephalitis consecutive to the meningeal affection. Furthermore, the softening of the ependyma and of the walls of the ventricles is now considered to be largely inflammatory, and not a mere result of imbibition.

The final step, arising out of Koch's discovery, was that the disease is in all cases caused by the invasion of the tubercle bacillus; either by local infection through lymphatic paths of which the subarachnoid space may be regarded as one, or, more commonly, through the blood-stream in embolic fashion along the distribution of the middle cerebral arteries.

Causes.—Tuberculous meningitis is a disease of childhood rather than of adult life. It may occur, however, at any age, though with rapidly lessening incidence as years advance.

Children at the period of the first dentition seem to be particularly liable to it. Of 100 cases, collected and analysed by Dr. Still, occurring in children under twelve years of age, in which the disease was verified by necropsy, 48 were under two years old: 83 of the 100 cases occurred during the first five years.

During the first twelve months of life non-tuberculous forms of meningitis are more frequently met with than the tuberculous, but in the accompanying table it will be seen that ten cases of the latter disease were recorded. The youngest patient was aged three months; but some cases have been reported even in younger patients than this. It is generally held that after puberty many more females are attacked by tubercle than males; but, so far as tuberculous meningitis in adults is concerned, the careful statistics of Seitz indicate that more men are attacked than women.

Incidence according to Ages in 100 Cases under 12 Years of Age. (Collected by Dr. Still.)

Under 6 Months.	6 Months-1 Year.	1-2 Years.	2-3 Years.	3-4 Years.	4-5 Years.	5-6 Years.	6-7 Years.	7-8 Years.	8-9 Years.	9-10 Years.	10-12 Years.
3	10	35	13	13	9	5	4	4	2	1	1

Incidence according to Age in 126 Cases between 16 and 60 Years of Age. (Seitz.)

16-20 Years.	21-25 Years.	26-30 Years.	31-35 Years.	36-40 Years.	41-45 Years.	46-50 Years.	51-55 Years.	56-60 Years.
28	29	23	14	13	11	4	3	1

Tuberculous meningitis has been observed repeatedly in two or more children of the same family, and likewise in families of which other members have suffered from divers manifestations of tubercle. But the absence of a tuberculous family history is a fatally delusive argument against the recognition of early symptoms of tuberculous meningitis. In a large number of cases a single child out of a large family may die from tuberculous meningitis, and no other member of the family show any indication of tubercle whatever. A family history of tuberculous disease was obtained by Dr. Still in 47 out of 100 cases (that is, nearly 50 per cent); but this proportion has no special significance, for almost exactly the same proportion can be obtained in the family history of persons not suffering from any tuberculous disease.

Insanity and hypochondriasis have been recorded in the family history of some cases of tuberculous meningitis, especially of the adult cases; and it is well known that a large number of insane patients die of phthisis.

There is no build of body peculiar to tuberculous meningitis. The disease may attack either an adult or a child who is the subject of phthisis and presents therewith any degree of emaciation. On the other hand, children apparently well nourished, with a fair amount of subcutaneous fat, are frequently attacked. Fair bodily nutrition is quite consistent with latent tubercle.

The common opinion that a large head and especially a prominent forehead make a child prone to tuberculous meningitis is quite absurd. This was pointed out by Whytt long ago. The reason for the popular belief is probably that the large forehead indicates rickets, and that a rickety child is more liable to convulsions than a non-rickety one.

The trend of modern doctrine has been towards the hypothesis of the introduction of tuberculous virus from without, especially by way of food. From the experimental observations of animal tuberculosis it appears certain that tubercle can be induced in the lower animals by feeding them with tuberculous material; and it has been held that the relatively large amount of mesenteric and intestinal tuberculosis found in young children is due to taking tuberculous milk into the alimentary canal. This is said to form a focus from which the dissemination of general tuberculosis, and therewith tuberculous meningitis, may take place; but complete clinical evidence, so far as child life is concerned, is not yet forthcoming (*vide* "Report of Royal Commission on Tuberculosis").

It has hitherto been difficult to prove any direct relation to artificial feeding in the tuberculous meningitis of young infants; and it is certain that it may occur in infants still at the breast, even though there be no naked-eye evidence of tuberculosis of the mother's mammæ, and when she is apparently healthy. It is possible that in such a case there is some latent deposit of tubercle in the mother which an injection of tuberculin might make manifest, or which at some time after lactation might become active.

The **causation** of tuberculous meningitis is, strictly speaking, the causation of general or local tuberculosis plus the special invasion. It is rarely, if ever, primary: almost invariably some tuberculous focus of earlier date than the meningitis can be found. The focus may be a localised caseous process in any part of the body, as, for example, in the lungs, glands, bones, joints, skin, or generative organs.

It is a perfectly legitimate criticism of the recorded cases of tuberculous meningitis in which no other tuberculous focus was found in the body, that the examination was not sufficiently exhaustive. In many cases *tuberculosis of glands following simple enlargement from* some peripheral irritation, such as catarrh of mucous membranes, especially of the throat, skin irritation, or carious teeth, forms the starting-point of a meningeal tuberculosis.

In other cases the disease forms part of a general acute miliary tuberculosis; but here also a primary focus generally exists; and in a large number of cases a breaking down of caseous glands seems to be the source of general infection.

A small number of cases arise by direct extension from a tuberculous mass in the cortex of the brain; and a few are associated with a caseous process in the middle ear, or with some tuberculous caries of other cranial bones and of the vertebræ. In a case of Demme's tuberculous meningitis followed tuberculous ozæna. A curious relation, noted in two cases by Henoch, was the occurrence of tuberculous meningitis simultaneously with the abrupt subsidence of some enlarged cervical glands: with this may be compared a case, long under my own observation, in which the sudden retrocession of an extensive eruption of phlegmonous scrofulides coincided with the outbreak of fatal tuberculous meningitis. There are certain facts which seem to show that in very rare cases surgical interference with tuberculous lesions, by the stirring up of latent deposits, may lead to discharge of tuberculous material into the general circulation, and so induce a general tuberculosis of which meningitis is a part. Two examples may be given: the onset of tuberculous meningitis was observed within a few days after forcible straightening of a hip joint affected by subacute tuberculous disease; and, in the other case, within a few days after the removal of tuberculous glands from the neck. It is held by some surgeons that the scraping operations on tuberculous foci are attended with greater risk of entry of tuberculous material into the blood-stream than are complete excisions.

Tuberculous meningitis has been said to follow erythema nodosum; but from the reports it may fairly be asked whether, in some of these at any rate, the initial lesions were not tuberculous periostitis.

Of remote causes, the exanthems are the most important; especially measles and whooping-cough: the former precedes tuberculous meningitis, as also other forms of tuberculosis, with remarkable frequency. It is probable that the exanthems not only lay the foundation for tuberculosis, but also reawaken any latent deposit: it is noteworthy that many of the exanthems are associated with glandular enlargement.

Starvation and adverse circumstances probably play some part in the causation of this as of other forms of tuberculosis. Injury, especially a fall on the head, has often been assigned as a cause; it may act as a determinant.

Overpressure in education has also been alleged as a factor in the causation, but the evidence adduced is not satisfactory.

The immediate determining causes of the onset of tuberculous meningitis are very mysterious; it seems likely that any general lowering of nutrition, or an irritation in the presence of previously caseating glands, by calling tuberculous foci into fresh activity, may determine an attack of the disease; but it is often difficult to prove any such sequence.

Symptoms.—The onset of tuberculous meningitis is either insidious or abrupt. It is insidious in those in whom some latent local tubercle has been consistent with moderately good health; it is abrupt in those whom advanced tuberculous disease has brought into a more or less emaciated condition.

The insidious onset is the more common of the two, and constitutes what is called the prodromal stage. As this is met with in children it may last from two weeks to three months, and is ill-defined throughout. The child seems out of sorts, he lies about instead of playing with his toys, he sleeps badly, he grinds his teeth and may have night terrors. His appetite fails, he loses flesh, and occasionally he vomits, especially at night, and without apparent cause. At this stage there may be a very fœtid smell of the breath, as if from gastric catarrh. The bowels are generally constipated. There is nothing uniform about the temperature. The dominant features are languor and irritability, the latter being manifested when the child is disturbed. In children of the school age change of temper is sometimes observed: a child previously obedient and amiable becomes irritable or wilful. He sometimes complains of headache, often frowns, and occasionally flushes. In a child over five years of age mistakes in speech may be noticed: the speech may be slow and very deliberate, as of laboured production; a marked reluctance to answer questions may be observed, and sometimes even a taciturnity amounting to speechlessness for days and weeks; although when questions are asked, the child may answer them. In adults, speech alterations often occur. There may be true aphasia, or that delay in answering questions which suggests cerebral inertia. A boy, aged about eight years, in the prodromal stage of what proved to be tuberculous meningitis, was sent to a shop to buy something. He found when he got to the shop that he was unable to ask for what he wanted. A very young child does not generally make mistakes of speech, but is often speechless for a day or two at a time, takes no notice of mother or nurse, asks for nothing, drinks only when liquid is presented, and sometimes silently passes urine in its bed, contrary to its wont.

The patient often stares and frowns at the doctor, but presents no other oddity of manner. It is most important to remember that in adults the onset of tuberculous meningitis is sometimes indistinguishable from that of hysteria; and the suppression of such initial symptoms by douching and electrical applications may give rise to delusive hopes. Incontinence of urine and fæces, again, associated with strange behaviour, occurring in the prodromal stage in adults, may likewise be set down to hysteria; and many cases of tuberculous meningitis in adults have at the onset been mistaken for early mental disease.

Delirium is rare in children, but not uncommon in case of adults; and *in some instances of early tuberculous meningitis even delirium tremens* is simulated. The early headache is much more severe in adults than in children. Lethargy, like that in the early days of a severe exanthem such as typhus, has been recorded; in such cases there may be prolonged retention of urine.

Children are more somnolent than adults. When the disease supervenes on some chronic tuberculous illness which has confined the patient to bed, the onset of tuberculous meningitis may be unsuspected, until a squint suddenly appears, or a complaint of double vision, or a slight facial or upper limb palsy.

Another mode of sudden onset in tuberculous meningitis in bedridden patients is capricious vomiting, apart from cough.

Finally it is to be noted that the supervention of tuberculous meningitis on severe or extensive tuberculous disease elsewhere is sometimes quite latent, and not discovered till after death.

In considering the prodroma generally it will be seen that although no one of these symptoms singly is pathognomonic, yet, in reviewing a case from beginning to end, their cumulative value is very great. The prodroma probably correspond with some dissemination of tubercle; and it is a matter of experience that during this vague period some slight chronic enlargement of glands in the neck or under the jaw, or some impaired resonance near the manubrium, although unattended with active symptoms, may give a cue to the imminent disease. The invasion of tuberculous meningitis Dr. Gee defines as the period at which such new symptoms occur as enable us to pass from the uncertainties of the prodromal stage, and to declare positively that, from this date at all events, the child has sickened with hydrocephalus. But it is fair to say that these new symptoms are often no more than exaggerations of one or more of the prodroma—such as vomiting, headache, or lethargy associated with constipation. Of definite new manifestations the most striking invasion-symptom, though not the most frequent, is convulsion. Such invasion-convulsion, when succeeding the prodroma, is in most cases general or bilateral; it is either a generalised rigidity with pallor and loss of consciousness, or it is divided into tonic and clonic stages affecting both sides of the body alike. From this period the disease has been divided into three stages, namely (*a*) the stage of irritation—referred to the meninges and cortex; (*b*) the stage of pressure—referred to the ventricular effusion; (*c*) the stage of relaxation and paralysis—referred to extension to the medulla oblongata (Traube).

But the symptoms of the first stage overlap those of the second, and the most satisfactory method is to consider each symptom separately as follows:—

Facies.—In the earliest stage the face shows nothing more than the frowning; but after the invasion there is often at times a vacant stare, with pupils dilated, and the eyes fixed on some distant object—"the far-off look," as it has been called. The flush of the cheeks, which in the prodromal stage was occasional, becomes more persistent, and is sometimes accompanied by a scarlet suffusion over the rest of the body, and a blotchy erythema of the palms. The tache cérébrale, or red flush elicited by stroking the skin, though by no means peculiar to this disease, is very frequently obtainable. In some cases a white streak appears before the flush. In the late stage any part of the body flushes if subjected to pressure.

During the prodromal and early stage of the disease the *skin* is dry and harsh; but in the last forty-eight hours it is often bathed with sweat.

Decubitus is very characteristic: quite early in the disease the child lies persistently on one side; if turned on its back or face it turns back on its side at once and lies with its knees flexed on the abdomen, and usually with the elbows flexed and drawn to the sides. It resents any interference; often, if disturbed, it will cry out peevishly, "Go away"; and if the bed-clothes be pulled back it will often clutch at them to draw them back (Stocker's sign).

In the later stage, when paralysis and coma have supervened, the child often lies on its back, low down in the bed, with the arms and legs extended, and the hands lying one over the other just above the pubes.

Photophobia is more common in adults than in children. The *hydrocephalic cry*—a sudden shrill and apparently causeless scream—is characteristic of intracranial disease, but not especially of tuberculous meningitis; the majority of cases run their course without its occurrence. Usually as long as the child is undisturbed it lies quite quiet in the position described; but occasionally there is great restlessness, the child rolling itself from side to side with such violence as to require a padded bed. In rare cases there is delirium, which may be quiet and muttering or wild and noisy. The occasional simulation of delirium tremens in adults has been mentioned already.

Headache is generally present in adults; but children often make no complaint of it, though the raising of the hands to the head, or the boring of the head into the pillow, may betray its presence: in some cases, however, the child cries out frequently, "My head, my head," and some of the sudden screams may be due to paroxysms of headache. Headache in adults is more commonly in the frontal region; but in children generally is not localised.

Pain is referred sometimes to other parts of the body, as to the abdomen, to the soft parts of the limbs, to the joints, and to the spine. Complaint of pain in the knees was so severe in the early stage of one case as to suggest rheumatism, and the application of cotton-wool round the joints. In a girl, aged ten years, complaint of the spine and well-marked local hyperæsthesia, in the absence of any other symptoms except pyrexia, were set down to hysteria; but within a few hours coma supervened, and at the necropsy tuberculous meningitis was found.

Vomiting, although one of the most frequent invasion-symptoms, is singularly variable in the subsequent stages of the disease. In rare cases it is incessant for days together; more commonly it recurs once or twice in the twenty-four hours. It is of the cerebral type; the contents of the stomach seem to regurgitate without retching or effort. Often no

Symptoms.—In a well-marked case of progressive muscular atrophy the first thing that a patient complains of is some weakness of certain muscles of the upper limb of one side; the muscles most frequently attacked are the small muscles of the thumb, and the first movements to be affected are the delicate and complicated movements of picking up a pin or fastening a button. Pain and all affections of sensation—such as tingling, numbness, and anæsthesia—are, as a rule, absent. The difficulty of performing certain movements directs the patient's attention to the thumb, and very soon he notices a distinct flattening of the thenar eminence.

On examination there is found some wasting of the small muscles of the thumb, with weakness and defect of two principal movements, namely, abduction of the thumb and opposition of the thumb: the former is best tested by laying the hand flat on the table with the palm upwards, and making the patient move the thumb upwards away from the palm. By opposition we mean the power of touching the tips of all the fingers in succession with the tip of the thumb, and more effort in opposition is required in the case of the index finger than of the fourth finger. As a rule it is not possible to detect the order in which the different small muscles are attacked, but the two movements mentioned above cannot be performed by the long flexors and extensors of the thumb in the absence of the small muscles. Again, flexion of the metacarpo-carpal joint cannot be performed by the long flexor alone, unaided by the small muscles, without antecedent flexion of the other joints of the thumb.

The first interosseus muscle is usually next attacked; its weak action is shown by an inability to abduct the first finger from the middle finger, and its wasting by the absence of the muscular swelling on the dorsal aspect when the patient presses the thumb firmly against the metacarpal bone of the index finger. Defects of the other interossei are shown by the difficulty of separating or abducting, and adducting each finger when the palm is placed flat on the table (as it is not possible to separate the fingers when they are flexed at the metacarpo-phalangeal joints), and by the hollowness of the interosseal spaces. Wasting of the hypothenar eminence and any inability to abduct the little finger indicate the extension of the disease to the small muscles of the little finger. One hand is usually affected before the other, and with about equal frequency on the two sides; but in a few months the other hand becomes involved in a similar manner. The disease is always bilateral, and generally symmetrical.

Before the small muscles of the hand are completely paralysed the flexors and extensors of the forearm usually become implicated; but if this be not the case, the hand assumes a peculiar clawed shape—the "main en griffe." This state, which was first explained by Duchenne, is due to the loss of power of the interossei, whose action is to flex the metacarpo-phalangeal joints of the fingers and to extend the phalangeal joints; hence when the interossei are paralysed there is flexion of the phalangeal, and hyperextension of the metacarpo-phalangeal joints of the fingers. Another deformity in extreme cases is that the thumb becomes so rotated outward that the palmar surface of the thumb looks the same way as that of the fingers; while, owing to the atrophy of the lumbricales, the flexor tendons can be seen in the palm of the hand.

After the muscles of the hand the muscles of the forearm are usually the next to be affected, and, as a rule, the flexors before the extensors, and the flexors of the fingers and thumb before the flexors of the wrist; on the other hand, the extensors of the fingers and thumb are usually affected before the extensors of the wrist.

In the upper part of the arm the muscle which is usually next to be affected is the deltoid, the rounded contour of the shoulder being lost and the head of the humerus being felt; after that follow the biceps, the brachialis anticus, and then the supinator longus; the supra- and infra-spinati are usually affected at the same time as the deltoid, and the teres major and minor and subscapularis also become involved. The serratus magnus is often attacked, and if the deltoid be strong enough to advance the humerus to the horizontal line, the posterior border of the scapula projects like a wing, owing to paralysis of the serratus. The rhomboids and the middle and lower part of the trapezius are often wasted, but the highest part of the trapezius between the occipital bone and the clavicle always escapes. There are three other muscles which nearly always escape; namely, the triceps, latissimus dorsi, and the lower half of the pectoralis major. The upper (clavicular) half of the pectoralis major is usually involved along with the deltoid, and in this case the action of advancing the humerus to the horizontal line is lost; but the upper part of the pectoralis may still retain its power of acting with its lower (sternal) half in adducting the humerus,—thereby giving an instance of paralysis of a muscle of a limb, from a spinal cord lesion, for one form of movement, but not for another.

The condition of most of the above muscles can be ascertained by their wasted condition and failure to move the joints; but there are two muscles between which it is often difficult to make a diagnosis, namely, the trapezius and serratus; for a projecting scapula, when the arms are advanced, may occur with paralysis of the middle part of the trapezius, or with that of the serratus. When the deformity is due to paralysis of the trapezius, no fibres can be brought out by direct faradisation over the paralysed part of the muscle, and the patient is still able to draw the scapula forward, as for instance in pushing with the extended arm; moreover, the posterior border of the scapula projects when the humerus reaches the horizontal line, owing to the scapula not being kept applied to the chest wall by the trapezius, while above this line the deformity disappears as the lower end of the scapula is rotated forwards by the serratus; whereas in paralysis of the serratus magnus, the separate movements of elevation of the advanced humerus above the horizontal line, and of drawing forwards of the scapula, as in pushing, are not possible, and the faradic reaction of the serratus muscle may not be obtained. This last defect, however, is an uncertain symptom, as it is difficult, especially in stout people, to faradise the muscle even in health.

The muscles of the neck, especially the extensors, are liable to be affected; and in extreme cases the head falls forward like a dead weight, and can only be extended by the patient jerking his head backwards, so as to get the centre of gravity of the head behind the spine; the sterno-mastoids are not affected as a rule, but they may be.

Of the other trunk muscles those of respiration are the most important. The most common condition is a palsy of the intercostals, when the respiration is purely diaphragmatic, but in some cases the reverse is the case; the abdominal muscles, such as the rectus abdominis, are affected less frequently.

The legs very often escape altogether, but in some cases wasting with loss of power begins in the anterior tibial muscles before the hands, and it may involve the extensors of the knees and gluteal muscles.

Cases which begin in the lower limbs sometimes run a very rapid course, as in the case of a man, aged 47, who, after a fall with his horse, began two or three months later to have difficulty in walking, and five months later weakness of the hands. The wasting, which was symmetrical, began in the anterior tibial muscles and the small muscles of the feet, followed soon after by that of the small hand muscles; the first loss of movement was that of dorso-flexure of the ankles, the right preceding the left foot, and eversion of the ankle being lost before inversion. In about two months more (eight months from onset) no contraction of the tibialis anticus was obtained to faradism, though the muscle still reacted slowly to a strong constant current, and KCC > ACC. Ten months from the onset the gastrocnemii and solei were wasting and getting weak, and also the thenar and hypothenar muscles. Fifteen months from onset he had no power to flex the ankle or to extend the toes; he could hardly contract the calf muscles, and he had lost all power over the small thumb muscles. At 18 months the thigh muscles began to waste, and the knee-jerks disappeared. At 21 months the diaphragm was not acting on either side; and, though the thigh muscles and the upper and lower arm muscles acted well, there were marked fibrillar contractions of the extensor cruris and the pectorals. The patient died two years from the first appearance of the symptoms. The electrical reactions of the muscles presented a steady diminution to the faradic current, till there was no reaction to the strongest current; to the galvanic the reaction became less, and the current had to be increased from 3 milliampères to 11; although the action became sluggish it was not always better to the anode than to the cathode closure. A necropsy was not obtained, but the fibrillar tremors, the electrical changes, and the absence of any form of anæsthesia or of pain made it extremely probable that the disease was progressive spinal muscular atrophy beginning in the anterior tibial muscles.

The muscles supplied by some of the cranial motor nerves are liable to be affected; this condition constitutes *bulbar paralysis*—a disease which occurs sometimes alone, as well as in association with progressive muscular atrophy; it will be described separately (p. 223). The changes in the motor cells of the medulla are, however, the same as in the anterior horns of the spinal cord.

Associated with atrophy and paralysis of the hand muscles, contraction of the pupil on the side most affected is sometimes observed, with loss of dilatation to shade; this is due to the implication of the dilator fibres of the iris which come off from the second dorsal root to join the sympathetic.

The electrical changes in the muscles are of great importance, and are especially useful in distinguishing these cases from the myopathies. The most important changes found are in the muscles rather than the nerves. The nerves react normally to the faradic current, and also to the galvanic current, so long as there are any fibres left to respond, but the amount of contraction produced is diminished; whereas the muscles which are wasted, while reacting through their nerves to the faradic current, show the reaction of degeneration with the constant current by giving a slow contraction, and by reacting better to the anodal (positive) closure than to the cathodal (negative) closure. This is called *the partial reaction of degeneration.* In some cases, although the contraction be slow, the cathodal closure is equal to or even greater than the anodal closure; and the slow, deliberate contraction of a muscle is more often an indication of degeneration than increased reaction to the anode. After a time the faradic reaction is lost in the nerve going to the muscle, and still later the reaction of the muscle itself to the constant current is not obtained.

Another symptom of much importance is *fibrillar contraction.* On watching a muscle which is beginning to suffer, a slight flicker may be noticed in the direction of the muscle fibres. This is due to the contraction of individual fibrils, which start up under the skin, subsiding again immediately. The flickerings are increased by mechanical irritation, as by tapping the muscle, or when the muscles are fatigued. They do not occur in those muscles which are too much wasted to have any fibres left to contract. They are probably due to a hyper-excitability of the degenerating cells in the anterior horns of the spinal cord. In some cases the fibrillar contractions are so strong as to flex or extend one finger involuntarily. Besides the fibrillation, *the reaction of the muscle to direct percussion* over its motor point is more readily obtained than in health.

In pure cases of progressive muscular atrophy there is no rigidity, and when the muscles, say of the forearm, are completely wasted, the wrist-joint is quite flaccid; the only rigidity there may be is due to wasting of muscles, such as the flexors of the wrist and fingers, while their antagonists, the extensors, are not affected, so that the joint is drawn into a fixed position: yet even here the rigidity is not so marked as in cases of infantile paralysis.

The deep *reflexes*, such as the knee-jerk, are not altered, unless the extensors of the knee be involved in the wasting, when the knee-jerk is diminished and finally disappears; but ankle clonus is not obtained, and the radius tap is not increased.

The superficial reflexes, such as the plantar, are lost if the muscles which should respond to the stimulus are much degenerated.

Sensation is never affected in any way, and pain is usually completely absent.

The sphincter vesicæ and sphincter ani are not affected, or very rarely so, but sexual power is sometimes lost.

The *course of the disease* is gradually progressive, and, as one side begins to waste before the other, the time may vary from a few weeks to as much as a year before the other side is affected. The disease may become stationary at any period, so that the wasting may progress for two or three years, and then become arrested. The wasting of the legs, if it occur, comes on as a rule after the arms.

If the limbs alone are involved there is no danger to life; but when the trunk muscles, and especially the muscles of respiration, are affected, there is danger of death from pneumonia and bronchitis; and when the bulbar nuclei are involved, the patient is liable to death from choking or from cessation of respiration.

Although the above description is that of the disease as most commonly met with, anomalous cases occur, of which the following is an example:—

A curious case of a child 15 months old has been recorded by Werding, in which there was gradual atrophy with paresis of the muscles of the back, the gluteal region, and the quadriceps cruris; then of the neck and throat, the thigh, the upper arm, forearm, and leg, and, lastly, of the hand and foot muscles. The atrophy was "en masse," and there was no hypertrophy. Bulbar symptoms occurred with fibrillar twitchings of the tongue. There were flaccid paralysis, electrical reaction of degeneration of the muscles, and loss of the knee-jerks; lordosis of lumbar spine was present; sensibility and the sphincters were normal. At the autopsy there was found a primary disease of the anterior horn cells without a trace of inflammatory appearances, marked degeneration of the anterior roots, and simple atrophy of the muscles.

The case is remarkable as involving the back muscles first and the hand muscles last, and also as occurring in a patient so young. A somewhat similar case was previously published by Werding.

As stated on page 208, heredity is not usually noted in progressive spinal muscular atrophy, but J. Hoffmann has brought forward five cases in members of one family, and two in another. The patients were between the ages of one and five years, and were afflicted with atrophy and paralysis beginning in the back, in the pelvis, and in the thigh muscles; later the shoulder and arm were affected, and last the fingers and the toes. The atrophy was quite symmetrical, the sphincters were not affected, but the tendon reflexes were lost. There was no pain, no fibrillar tremors, but the electrical reactions of degeneration were well marked. The parents and grandparents were healthy. At the autopsy on one case were found atrophy and shrinking of the anterior horn cells, atrophy of the anterior roots and of the peripheral nerves, and simple atrophy of the muscles. The case is of importance in showing that spinal muscular atrophies depending on lesions of the anterior horns may begin in the trunk muscles and affect the hand muscles last; and that the occurrence of muscular atrophy in several members of the same family is not an exclusive habit of myopathies.

Another rather similar case has been reported by Strümpell, in which progressive muscular atrophy began in the hands of a man aged 40, and spread up the arm to the shoulders and back muscles. There were no hypertrophy, no fibrillar twitchings, and no reaction of degeneration. The mother of the patient had the same disease. At the necropsy were found simple atrophy of muscles and degeneration of the peripheral nerves and of the anterior horn cells. There is, therefore, in this case also a history of heredity, and at the same time no fibrillar tremors and no reaction of degeneration, symptoms characteristic of myopathies, and yet the disease was in the anterior horn cells. Strümpell regarded the case as a myopathy, and the atrophy of the muscles as a simple one, and not to be explained by a descending degeneration from the anterior horn; as though the atrophy had begun in the muscle, and then by gradual ascent the peripheral nerve and the anterior horn cell had become affected secondarily. He thinks that between spinal and myopathic muscular atrophies a hard and fast line cannot be drawn, and that it is doubtful whether there is such a disease as an acquired progressive atrophy.

Attacks have been described, coming on rapidly in a few days or a few hours with loss of power in the extensors of the wrist and fingers, and occurring first on one side and soon afterwards in the other (Gowers, *loc. cit.* p. 483), as an occasional mode of onset in progressive muscular atrophy, and followed by gradual wasting of the other muscles of the limbs.

Morbid anatomy.—The essential pathological change is in the large cells in the anterior horns of the spinal cord, and especially in those of the cervical enlargement. Under the microscope the gray matter of the anterior horns is wasted and paler than normal, and the large cells are atrophied and shrunk; many have lost their processes, while in some sections the cells have altogether disappeared. If the muscles of the legs are wasted the lumbar enlargement will be similarly affected. The interstitial tissue is altered, so that with the atrophy of the finest nerve-fibres there is an overgrowth of connective tissue with increase of connective tissue cells. The anterior roots which come off from the affected sections are diminished in size and degenerated.

The lateral columns in pure cases are not affected.

Whether in cases of progressive muscular atrophy the lateral columns are affected or not has been a matter of much discussion, especially by v. Leyden and Sir W. Gowers. The latter author states that he has "not yet met with a single case of progressive muscular atrophy in which the pyramidal tracts were unaffected," and further on (*loc. cit.* p. 494) "that the assumption that the primary lesion is the degeneration of the pyramidal tracts, and that the affection of the gray matter is secondary even where the atrophy is atonic, is unwarranted as far as the atonic atrophy is concerned." He also thinks that the pyramidal tracts are degenerated, if not constantly, at any rate in such a very large proportion of cases that we are in effect giving a new name to an old disease.

With the above opinions I regret that I am unable to agree, as there is no doubt in my own mind that cases are met with of pure atrophy of the anterior horns without any change in the lateral columns, and such a case of a typical progressive muscular atrophy I have had lately under my care. This patient began with atrophy of the small hand muscles and later of the shoulder muscles, the legs being but slightly affected, and never at any period was there any rigidity or any increase of the deep reflexes; after death atrophy of the cells of the anterior horns was found, but no change in the lateral columns. The question has also been taken up by J. B. Charcot, who brings forward two cases of pure progressive muscular atrophy, with atrophy of the anterior horns and slight degeneration in the anterior ground bundles of the anterior columns. I still, therefore, hold the opinion that it is possible either to have a pure atrophy of the anterior horn cell and its prolongation down to the muscle, forming the lower segment of the motor tract, or to have only the upper segment affected, as in the early stage of amyotrophic lateral sclerosis; or, again, to have both segments involved, producing the deuteropathic form of this disease.

Déjerine has also published two cases of progressive muscular atrophy in which the anterior horns were affected and the white matter was normal.

The peripheral nerves in the limbs contain many degenerated nerve-fibres.

The muscles are very much wasted, and, in extreme cases, can hardly be distinguished from the surrounding fat; but in slight cases they are only pale. Under the microscope the transverse striation is seen to be lost; the muscle first becomes granular and then undergoes fatty degeneration.

According to Hayem, there is a simple atrophy of the muscular fibre with retention of the cross striation, which persists to the end. This atrophy is accompanied by proliferation of the cell elements of the sarcolemma, which distend its sheath, and the muscular substance then becomes segmented; the newly-formed cell elements tend to atrophy, and the muscular substance goes on dividing, and sometimes disappears altogether without showing any trace of granular fatty degeneration. The perimysium undergoes changes, and a deposit of fat may occur, making it difficult to recognise the atrophy of the muscles. The muscle-spindles have been found unaltered by several observers.

Pathogeny.—The order in which the muscles are involved and their grouping is of great importance, especially in the matter of diagnosis. In a typical case the atrophy begins in the small muscles of the thumb, and it is noticed that all the muscles suffer equally. There is no distinction between those supplied by the median or ulnar nerves. After the interossei and the small finger muscles, the disease, as a rule, attacks the flexors of the fingers, and then the flexors of the wrist before the extensors of the fingers, thumb, and wrist. Almost simultaneously with the thumb muscles, or soon after them, the deltoid begins to waste, and is followed by the supra- and infra-spinati, the biceps, the brachialis anticus, and the supinator longus. The last muscles to be affected are the latissimus dorsi, the lower half of the pectoralis major, and the triceps. The probable explanation of this is that the muscles are attacked in the way in which they are integrated together in the brachial enlargement of the spinal cord. Different tables of the arrangement of the muscles have been given by various observers, but the list which has been made out by Thorburn from fractures of the spine is one of the most trustworthy, as regards man. According to Thorburn, the biceps, deltoid, and supinator longus are supplied by the 5th cervical; the subscapularis, teres major, latissimus dorsi, pectoralis major, triceps, serratus magnus also by the 5th, and pronators by the 6th; the extensors of the wrist by the 7th; the flexors of the wrist by the 8th; and the intrinsic muscles of the hand by the 1st dorsal. It will thus be seen that progressive muscular atrophy begins in the brachial enlargement of the cord, at the level corresponding either to the 1st dorsal or to the 5th cervical—either at the lower or the upper end of the enlargement; while the part corresponding to the 6th cervical, and supplying the triceps, latissimus dorsi, and pectoralis major, escapes. The combination of the deltoid, biceps, and supinator longus in spinal cord and root lesions has long been shown by Erb, but the combination of the latissimus dorsi, triceps, and the lower half of the pectoralis major, I believe I was the first to show, in a case of infantile paralysis, in 1885.

As will be seen under diagnosis, the grouping of the affected muscles is of the greatest use in determining whether the atrophy is idiopathic or due to a spinal lesion. What the cause is of the changes in the cells of the anterior horns it is very difficult to say; it seems to be a simple degenerative process, but whether it depends on vascular or nutritional changes it is not possible to speak definitively.

Diagnosis.—Cases of progressive muscular atrophy have to be diagnosed from several other forms of atrophy of muscles, and the most important points are:—that in progressive muscular atrophy the onset is very gradual; wasting does not follow the loss of power, but accompanies it; the muscles attacked in sequence and in grouping correspond, as a rule, to the order given above; fibrillar twitchings are present; the muscles give to electrical testing the reaction of degeneration, which is frequently partial; there is very slight pain or none, and no loss of sensation in any form.

Beginning with diseases which affect the muscles themselves, progressive spinal muscular atrophy differs from idiopathic muscular atrophy by the absence of enlargement in the distribution of the muscles affected, and by the presence of the fibrillar tremors, and the reaction of degeneration, which symptoms being absent in the latter disease. Neuritis has a more rapid onset, is attended with severe pain and tenderness of the muscles or nerve-trunks, and has a distribution corresponding, as a rule, to the supply of a nerve-trunk; moreover fibrillar tremors are absent, and the electrical reaction of degeneration is more complete, while on the sensory side tingling and anæsthesia occur. In neuritis of the cervical roots, or in pachymeningitis, the diagnosis is more difficult unless the paralysis follow an injury, as the distribution may be the same as in progressive muscular atrophy; but the paralysis comes on more rapidly in neuritis, and is attended by pain and anæsthesia and painful rigidity, though loss of sensation may be very slight.

In one rare case which I saw under Dr. Ringer (15) there was a gradual paralysis, which began in the hand muscles and extended up the arm, but affected the triceps before the biceps; it was attended by severe pain without any anæsthesia, and was due to a malignant growth which extended up the anterior surface of the spinal cord and involved the anterior and not the posterior roots.

Among other diseases of the cord itself, progressive muscular atrophy differs from acute anterior poliomyelitis in which the onset is sudden with loss of power, followed by wasting and loss of electrical reactions; and from syringomyelia where, though the onset is gradual and begins with atrophy of the hand muscles, sensation is much affected, sensibility to pain, heat, and cold being usually lost, while tactile sensation is retained; trophic changes in the skin, nails, and joints are also present in syringomyelia.

Cases of progressive muscular atrophy have also to be diagnosed from a peculiar form of atrophy which was described by Charcot and Marie in 1886, and independently by Dr. Howard Tooth later in the same year, who named it the peroneal type of progressive muscular atrophy. It seems that the condition described by J. Hoffmann (13*a*) as "Progressive neurotischen Muskelatrophie" is the same disease. The peroneal type is occasionally hereditary, it attacks several members of the same family, beginning in childhood with atrophy of the peronei, extensor communis digitorum, or extensor longus pollicis, or in the small muscles of the foot, causing equino-varus; and later the calf and thigh muscles are involved. Later still the small hand muscles waste, causing a claw-hand, and this is followed by wasting of the extensors and flexors of the wrist and digits—the supinator longus and the arm and shoulder muscles not being affected. Fibrillar tremors and the reaction of degeneration are present. Sensation is definitely affected over the parts which are paralysed, and pains occur. The knee-jerk is usually lost, and vaso-motor lesions are seen. Dr. Tooth suggested that the lesion is probably in the peripheral nerves. Autopsies have been made by Hoffmann, Eulenburg, Dubreuihl, and by Marinesco, in which the anterior horns were found intact; but there was neuritis of the peripheral nerves extending up to the posterior roots and sclerosis of the posterior columns of the cord.

The diagnosis from progressive spinal muscular atrophy would be made by the age and the presence of pain and anæsthesia.

Prognosis.—This is, unfortunately, not very favourable, either as regards arrest of the disease or restoration of the paralysed and wasted muscles.

The atrophy may become arrested either spontaneously or as the result of treatment; but as long as the disease is progressing it is not possible to say to what extent it will spread. According to Sir W. Gowers, the prospect of arrest seems to be greater in the cases in which the wasting is strictly symmetrical and nearly simultaneous on the two sides. If the disease is once arrested it often does not advance again; but in some cases, after apparently ceasing, fresh muscles are attacked, and this goes on till the death of the patient.

There is no danger to life as long as the wasting is confined to the limbs, but as soon as the thoracic muscles are attacked, or bulbar symptoms appear, there is great risk of death from intercurrent bronchitis or pneumonia, from choking, or from implication of the respiratory muscles. Two patients I have seen died in two years' time from paralysis of the respiratory muscles.

With regard to the recovery of the wasted muscles it is possible, but not probable, that nutrition in a muscle recently attacked may be restored to some extent; but in the case of a muscle which is so much damaged that there is no reaction to either form of electric current, no improvement can be looked for.

Treatment.—The general treatment for progressive muscular atrophy is to keep up the general health of the patient by fresh air, either in the country or the seaside; and, if the patient is fit for it, a sea-voyage is to be recommended. Exercise should be taken in moderation, but should always fall short of tiring the patient.

Local treatment has not much effect on the wasted muscles. Electrical treatment has been used very largely, and, though the results are of doubtful value, it is at any rate a means of exercising the muscles without tiring the patient. It is best to employ the constant current with the positive pole to the back of the neck, and the negative gently brushed over the affected muscles, being careful to use the weakest current which will cause a contraction.

Massage and rubbing have been frequently employed; this treatment seems likely to be useful, yet it often fails to arrest the wasting. With regard to internal remedies, cod-liver oil, quinine, and the syrup of iodide or phosphate of iron are requisite to keep up the general health. Of special nerve tonics arsenic and strychnine are useful. According to Sir W. Gowers, strychnine is most effective when given hypodermically, and succeeds thus after it has failed when given by the mouth. He recommends the nitrate of strychnine given once a day, beginning with gr. $\frac{1}{100}$ and rapidly going up to gr. $\frac{1}{40}$; when the malady is apparently arrested the injections are intermitted for one week in every three or four. It does not seem to make much difference whether the injection be given in the neighbourhood of the affected muscles or elsewhere. In cases where I have tried this treatment I cannot say that I have been very favourably impressed by the results.

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BULBAR PALSIES

These affections, as their name shows, are due to lesions of the medulla oblongata, or bulb.

The parts of the medulla especially concerned in these cases are the nuclei of the cranial nerves, which take their origin in the medulla, and especially those of the 7th, 11th, and 12th nerves, and the motor part of the 5th nerve, which takes its origin from the pons Varolii, and so much of the nerves themselves as lies within the medulla is involved also.

Besides the paralyses due to lesions of the nuclei and their nerves, certain affections of the tongue and throat are produced by lesions in the nervous system above the nuclei; and there are other palsies due to disease in the muscles themselves, which are quite distinct from disease of the nerves or their nuclei.

The movements of the tongue, of the larynx, of the soft palate, and of the pharynx are represented in the cortex cerebri, and for the most part bilaterally; thus a lesion of both sides of the cortex, or of both the internal capsules, or of both the pyramidal tracts above the nuclei supplying the muscles taking part in these movements, will produce palsies of them, very similar at first sight to the true bulbar palsies due to lesions of the nuclei or their nerves. The corresponding symptoms are called pseudo-bulbar paralysis, but, except for the purpose of diagnosis, this group does not enter into our consideration.

On the other hand, certain myopathies cause paralysis of the movements of the face, but these again are not instances of true bulbar paralysis.

The true bulbar palsies may be divided into nuclear and infra-nuclear and as some of them are acute and some are chronic, it will be advantageous to arrange them in the form of a table, describing the pseudo-bulbar forms, in which the lesion occurs in the cortex or the internal capsule or pyramidal tract of both sides, as supra-nuclear.

- Supra-nuclear, acute from lesion in cortex cerebri, internal capsule, pyramidal *tract, or in motor fibres just above nucleus.*
- Nuclear, acute, vascular
    - inflammatory
  - chronic, degenerative
  - tumours
- Infra-nuclear, within medulla, acute, softening
    - tumour
  - outside medulla, intra-cranial, meningitis, simple
        - syphilitic
      - tumours
      - neuritis
    - extra-cranial, tumours
      - cellulitis
      - caries of vertebræ
      - multiple neuritis.

From a clinical point of view it is better to describe all the acute bulbar palsies together, and to include in this group the lesions just above the nuclei, those affecting the nuclei themselves, and those just below the nucleus but inside the medulla.

The acute palsies may be subdivided into (A) those which are sudden, and (B) those which are rapid in their onset.

A. **Sudden bulbar paralysis**, called also apoplectiform.—The cause of these cases is always vascular; they may be due to a small hæmorrhage, but much oftener to thrombosis or embolism of one or more of the branches which come off from the basilar or vertebral arteries.

The occlusion in most cases is due to thrombosis occurring in syphilitic or atheromatous arteries. In rare cases embolism of the basilar may ensue from endocarditis, but the result of this would produce symptoms much more extensive than paralysis of the muscles supplied by the bulbar nuclei.

The affection usually occurs in middle or old age, but cases due to embolism or to syphilitic arteritis may be met with in younger people.

Symptoms.—The most important point of distinction between these cases and those caused by other lesions of the bulb is the mode of onset: this is quite sudden, with vomiting or giddiness, and with or without loss of consciousness; or the patient goes to bed apparently perfectly well and wakes up in the morning with bulbar symptoms.

In an extreme case there is paralysis of both sides of the tongue, of the soft palate, of the power of swallowing, and sometimes of the vocal cords. In other cases the symptoms are not so extensive, and may affect only one side.

In one case, which I saw with Sir Felix Semon and Mr. O'Connor, the patient, a man aged forty-two, went to bed quite well; on awaking in the morning he could not swallow, and fluids were coughed out of the larynx or returned through the nose; beside this there was paralysis of the levator palati on the right side, so that on phonation the palate was drawn to the left; the tongue was not affected, except that on protrusion it deviated slightly to the left, and the vocal cords acted normally. There was no anæsthesia, and the reflex action of the soft palate on the two sides, though deficient, was equal. In this case the sudden onset in the night was caused by a vascular lesion, probably thrombosis; but the exact position of the lesion was rather difficult to decide. It was observed that in the act of swallowing the larynx was raised up, and therefore that inability to swallow was due to failure of the constrictors of the pharynx; and, as it is very improbable that paralysis of one side of the pharynx would prevent swallowing, it is more likely that both sides were affected. We have, then, to find some part of the nervous system a lesion of which would cause paralysis of the right levator palati and both sides of the pharynx. The sudden onset in one attack was against the pseudo-bulbar form; for although the act of swallowing is certainly represented in the cortex cerebri at the lower end of the ascending frontal convolution, it is bilaterally represented, so that it would be necessary to have a lesion in the cortex of both sides, or in both internal capsules, to abolish the power of swallowing; besides, the action of the various muscles taking part in swallowing are co-ordinated in the cortex, and on stimulation of it swallowing occurs as one act, whereas in this case only part of the movement was deficient. It is not known whether the fibres from the pyramidal tract cross over to the "centre for swallowing" in the medulla as one bundle, but it is more probable that the fibres cross over separately to the different nuclei taking part in the action of swallowing. It would therefore seem possible in this case that these fibres from the pyramidal tract to the nuclei of the accessory nerve or of the glosso-pharyngeal nerve were affected just above the nuclei.

In another case which came under my notice there was a sudden onset with giddiness but without unconsciousness, followed by paralysis of the soft palate and of the vocal cord on the same side, and loss of sensibility to heat and cold and to pain of the opposite half of the trunk and limbs, but not of the face. This was probably due to slight thrombosis affecting the accessory nerve of one side on its way through the medulla, or its nucleus, and the sensory tract for the conduction of temperature and painful impressions for the opposite limbs and trunk, in the lemniscus above the decussation of its fibres.

In another case, a woman, aged thirty, suddenly had some loss of power and sensation of the left side of the body, and thickness of speech; on examination a few days later it was found that the power had been recovered in her limbs, but that she was suffering from "crossed anæsthesia." Over the left half of the trunk and the left arm and leg there was some tactile anæsthesia, which was not complete, with some loss of sensibility to pain and temperature; this area extended up the neck and up the posterior part of the left side of the head to the vertex, but it did not affect the left face. On the right side, over the area of the 5th nerve corresponding to the first and second divisions and to a less degree of the third division, there was marked deficiency to tactile sensations. There was slight conjugate nystagmus in all directions. A few days later she had weakness of the right temporal and masseter muscles supplied by the motor part of the 5th nerve, and also of the right facial muscles. She also had attacks of vomiting and giddiness, *with apparent revolution of objects from right to left.* As the patient recovered from the symptoms the seat of the lesion is a matter of conjecture, but it seems almost certain that it affected the nucleus or the fibres of the right 5th nerve on their way through the pons, thereby causing the anæsthesia of the right face and paralysis of the right muscles of mastication; and it also affected the lemniscus—the sensory tract—on the right side above its decussation, so as to involve the sensory fibres of the left half of the trunk and left limbs, while the sensory fibres to the left face escaped, their crossing being above the lesion. The affection of the right facial muscles was slight, and was perhaps due to extension of the disease to the fibres on their way to the facial nucleus from the opposite pyramidal tract. As she had weak cardiac action, the case was probably one of thrombosis of the branches of the basilar artery to the pons.

A case has been described by Senator in which a man, aged seventy-one, had a sudden attack of giddiness, vomiting, trouble in swallowing, and numbness in the left half of his face and left gums, and in both arms; the lower part of the left face was paretic, the tongue was protruded to the left, the legs were weak, the knee-jerks were absent, and there was deafness of the left ear. After death a softened patch was found on the floor of the left half of the 4th ventricle 7-8 mm. long by 4 mm. broad, and extending between the olive and the corpus restiforme. Recently Van Oordt has published a case of sudden onset in which the symptoms were inability to swallow, nasal speech, paralysis of the right half of the tongue, and of the whole of the muscles supplied by the right facial nerve, including the orbicularis oculi, and right hemianæsthesia. On examination there was found, with arterial sclerosis, a softening patch on the left side of the medulla at the level of the middle third of the inferior olive, and affecting the formatio reticularis.

The symptoms differ as the lesion is above the nuclei, in the nuclei, or involves the nerves coming off from the nuclei on their way through the medulla. Besides the grouping of the muscles affected, the condition of the nutrition of the muscles and their behaviour to electrical testing and to reflex irritation are very important.

The acute and sudden bulbar lesions which involve the nuclei, and the nerves which come off from them, differ in their symptoms from lesions above the nuclei in the distribution to the muscles, and in causing wasting of the muscles in addition to paralysis of their movements. It is difficult to make this out with regard to the soft palate and laryngeal muscles; but with the tongue, owing to the facility with which the tongue can be felt and seen, it is fairly easy to tell whether the mucous membrane is thrown into folds owing to the wasting of the muscular tissue. The muscles would also lose their reaction to faradisation, and would give the slow deliberate action to the constant current, reacting to the positive closure sooner than the negative—so that ACC is greater than KCC. The electric reactions of the tongue and the levator palati can readily be ascertained.

The reflex response of the soft palate to irritation of the fauces is also lost or very much diminished when the lesion is in the accessory nucleus or the nerve coming from it, so that if one nucleus is affected the palate is drawn up reflexly towards the non-paralysed side only, and not raised at all if both nuclei are involved.

The wasting of the muscles and the loss of reflex action are due either to the cutting off of the muscles from their trophic centre in the nuclei, or to destruction of this centre; it is often difficult therefore, if not impossible, to discriminate between the lesion of the nucleus and of the nerve coming from it. In the case of the hypoglossal nerve, however, owing to the proximity of the nerve to the pyramidal tract, there is more probability of some paralysis of the opposite limbs than if the lesion be in the nucleus; while the orbicularis oris is liable to be paralysed when the nucleus is affected, and not the nerve only.

The distribution of the muscles affected is that pertaining to the particular nucleus or nerve involved; but the muscles supplied by a bulbar nucleus do not always correspond to those by its nerve, as the latter may receive fibres from other nuclei before it leaves the medulla.

If there be no wasting of the muscles, and no electrical changes, and if reflex action be preserved, the lesion must be above the nuclei; but it is often very difficult to tell whether the lesion be in the bulb just above the nuclei, or higher up in the motor path. The mode of grouping of the muscles affected by supra-nuclear lesions is different from that of nuclear lesions, for as the cortex cerebri knows nothing of individual muscles, but is concerned with movements only (Hughlings Jackson), the paralysis which occurs from lesions of the cortex or pyramidal tracts of both sides will be that of complete co-ordinated movements; for instance, in lesions of both pyramidal tracts one would expect that all the muscles entering into the complex movement of the first stage of swallowing would be paralysed, whereas in a nuclear lesion it would be possible for some of the muscles to be paralysed and not others, the paralysis in one case having a physiological grouping, in the other an anatomical.

Of the difference of the grouping of the muscles affected in double cortical or pyramidal lesions, and in a single lesion involving the pyramidal fibres as they are decussating at the middle line to reach their nuclei, it is difficult to speak definitely, and theoretically the grouping of the latter should be physiological; but in the case already mentioned only some of the muscles of swallowing were affected; and this was also found in a case published by v. Leyden. With regard to double lesions, complete paralysis of the movements of mastication and of swallowing, of the vocal cords and of the tongue, cannot follow a lesion of the cortex or the motor tract, unless it be bilateral, for in the above movements the muscles of both sides are represented in each hemisphere: thus if one hemisphere is paralysed, the opposite hemisphere can carry on the work, but if both are affected, all voluntary movements are lost and reflex action only is retained. Hence in these cases there is a history of two or more attacks.

Cases of double hemiplegia producing these pseudo-bulbar symptoms have been described by Lepine, Jolly, Eisenlohr, Kirchhoff, Ross, Barlow; one of the most important being one by Barlow in which there was found on necropsy softening of the 2nd and 3rd frontal and ascending frontal convulsions of both sides, producing in the first attack right hemiplegia, followed in four months by a second attack of left hemiplegia, with symptoms of inability to show the teeth, to protrude the tongue or to talk, though he could understand signs; swallowing was difficult, and there was weakness of the limbs. Recently I have had a case in a woman in whom the two attacks were separated by an interval of four months: in the first attack the speech became thick, with weakness of the left side; in the second attack she lost all power of speaking and even of phonation, was unable to swallow, and the right arm was weak; two years after the first attack there was still complete paralysis of the soft palate on both sides to voluntary efforts, with preservation of reflex action and electric reactions. In this case the lesions were probably thrombotic and affected the anterior limb of the internal capsule of one side and, later, that of the opposite side.

Other cases have been described in which vascular lesions have been discovered in the internal capsule and lenticular nucleus of both sides; as in one described by Dr. Newton Pitt, in which the paralysis of the face and tongue, and of swallowing, was very complete.

In another case, published by Drs. Hughlings Jackson and Taylor, the patient had two attacks of hemiplegia affecting opposite sides, several years elapsing between the two attacks. The onset in the first attack was sudden, and articulation was lost for a week; in the second the left side was affected with a sudden onset, the patient could not protrude the tongue, and swallowing and speech were very difficult. The whole left side of the face was weak, the soft palate was completely paralysed, and the limbs of both sides were weak. Both pyramidal tracts were found degenerated and sclerosed.

The mode of onset in all these cases by two attacks, affecting first one side and then the other, and the affection of the limbs are symptoms of lesions seated in both motor paths rather than of a single lesion occurring at the point of decussation of the fibres from the motor paths just above the nuclei.

B. Acute inflammatory bulbar paralysis.—This second form, also included under the term "acute," has a rapid onset, but takes some hours or a few days to develop itself. The nature of the lesion is the same as that of acute poliomyelitis (infantile paralysis).

The condition is not at all common, and the changes depend on an inflammation involving the nuclei of the bulb.

Symptoms.—In one case, described by Etter, the attack began, in a boy aged fifteen, with general malaise, headache and vomiting, pyrexia and difficulty in swallowing; in the course of a week came on paralysis of both sides of the face, the soft palate, the tongue on both sides, but especially the left, and the left 6th nerve. After death from pneumonia, acute myelitis was found on both sides of the medulla in patches involving the left 6th nucleus, the left facial nerve within the medulla, the left hypoglossal nucleus, the right facial nucleus, the right hypoglossal fibres, the accessory nucleus of both sides, and below them the spinal cord as far down as the 4th cervical nerve.

The changes found are similar to those of acute poliomyelitis, and consist of dilated vessels with exudation of leucocytes, and destruction of the cells of the cranial nuclei; or, in less severe cases, the nerve-cells lose their processes and shrink.

Another case, described by W. Pasteur, occurred in a boy 2½ years old with a rapid onset of fever, diarrhœa, and sickness; the right facial muscles were completely paralysed with wasting, and there was difficulty of swallowing and paralysis with wasting of the right side of the tongue.

Vandervelde has published a case of an acute inflammatory attack with fever and headache, causing paralysis of the middle and lower part of the face, difficulty of swallowing, noises in the ears, and an inclination to fall to the right; at the autopsy were found leucocytes round the vessels and swollen axis-cylinders on both sides of the medulla.

Prognosis.—Acute bulbar paralyses are more favourable than the chronic forms, provided that the patient do not succumb at the time of the attack; or, in other words, if the attack be so slight that the patient do not lose consciousness, he will probably recover entirely, as in the case referred to on page 222; this is especially the case in people below fifty who have had syphilis. On the other hand, thrombosis, whether due to syphilitic or atheromatous arteries or to emboli, is very apt to recur, and a patient may have a succession of cerebral attacks, in one of which he may succumb. This is seen in pseudo-bulbar cases as well as in those of true bulbar origin.

If after an acute attack there be difficulty of swallowing and breathing, the prognosis is not so good; but probably these will diminish, and, if the patient get over the first week after his attack, his further improvement will depend on whether the lesion is above the nucleus, in the nucleus, or below it: the least favourable condition for future recovery is a lesion of the nucleus, it is better when the lesion is in the nerve within the medulla coming from the nucleus, and it is most favourable when the lesion affects the motor tract.

The *treatment* of acute bulbar attacks is different in the two varieties of sudden and of rapid onset. In the sudden form the cause of the vascular obstruction must be ascertained, and, in the absence of cardiac disease, Bright's disease, or atheromatous arteries, the treatment should be antisyphilitic, namely, inunction of mercurial ointment, followed by large doses of potassium iodide. Or if any one of the other diseases be present, the treatment will be modified accordingly.

In the acute inflammatory form the treatment should be that of acute poliomyelitis.

The great difficulty in most of these cases is the feeding; if there be any difficulty in swallowing, there should be no hesitation in feeding the patient by a soft tube passed through the nose.

Later, in nuclear and infra-nuclear cases, if the muscles are wasted, they should be treated by the constant current, and internally by quinine, iron and strychnia.

Chronic bulbar paralysis, chronic nuclear paralysis.—This affection, which occurs much more frequently than the acute forms, is also called "Glosso-labio-laryngeal paralysis."

The disease was first described by Duménil in 1859, and more fully by Duchenne in 1860; but before these a case had been seen and described by Trousseau in 1841, though apparently it was not published. In these cases atrophy of the spinal accessory and hypoglossal nerves was found, and it was remarked that the atrophy was identical with that found by Cruveilhier in progressive muscular atrophy.

Duménil evidently considered that his case, in which there was atrophy of the hypoglossal nerves, was a variety of progressive muscular atrophy, whereas Duchenne looked on the cases as quite different, for he says that in glosso-labio-laryngeal paralysis there is paralysis without atrophy, while progressive muscular atrophy is a lesion of muscular nutrition without paralysis; but as he describes the tongue in his cases as having very little motion and its surface as being a little wrinkled, there probably was atrophy of the tongue.

Causes.—The age at which the disease begins is usually between thirty and sixty, though a few rare cases have been observed much younger; for instance at eight years by Remak, and at twelve by Hoffmann (13).

Males are rather more affected than females.

In the family history some other form of nervous diseases is occasionally present, and, rarely, several members of one family have been affected.

Previous illnesses do not appear to cause the disease, nor does syphilis seem to enter into the causation.

Exposure to wet and cold and to severe mental and physical strain have appeared to be proximate causes in some cases, but for the most part the causes remain unknown.

Symptoms.—Chronic bulbar paralysis may exist by itself, but it is particularly associated with two diseases affecting other parts of the body. One of these is progressive muscular atrophy, and there is no doubt that chronic bulbar paralysis is the representative of this disease in the medulla—in fact, that they are one and the same disease occupying analogous parts of the nervous system; it is not strange, therefore, that the two forms should exist together. The other disease is amyotrophic lateral sclerosis, where the weakness of the muscles supplied by the medulla follows the symptoms of lateral sclerosis, and the weakness is in some cases followed by atrophy.

In chronic bulbar paralysis the symptoms are bilateral and come on, as a rule, gradually, with slight difficulty in speaking; but in one case I saw the difficulty was first noticed when the patient tried to sing, and after that the speech gradually became indistinct; it was probable that in this case the defect was not sufficient to affect the speech, but that the greater effort in singing brought it into prominence.

The tongue is usually the first organ to be attacked, and it shows itself in the difficulty of pronouncing certain consonants which are produced by the action of the tongue as it is approximated either to the teeth —dental consonants—or to the soft palate—guttural consonants. In the former case *t*, *d*, and *th* are difficult to pronounce and are first affected, and subsequently the consonants *k*, *g*, and *ch*.

Along with the difficulty of speaking, general weakness of both sides of the tongue is observed, with wasting of its muscular tissue. At first the tongue looks flatter and not quite so plump as natural, the movements become slower, and there is difficulty in protruding the tongue to the full extent beyond the teeth, and also in putting the tip into either cheek, or in elevating it. Early in the disease fibrillar contractions are observed in the form of fine linear quiverings taking place along the tongue in a longitudinal direction under the mucous membrane.

After the tongue the orbicularis oris is next involved, and it is noticed that the patient is unable to purse up the mouth, and loses the power of whistling (in trying this test the anterior nares should first be closed, as the soft palate is often unable to close the posterior nares), or of blowing out a candle. In the early stages the strength of the orbicularis oris may be tested by inserting the forefinger and thumb between the lips, and by trying to separate the finger and thumb against the pursed-up lips; in a healthy person this is not easy. At the same time the speech is still further affected, and especially the explosive labial sounds *p*, *b*, although the aspirate labial sound *f* and the resonant labial *m* can still be sounded, as in these two sounds the lips are not required to be tightly closed. As the paralysis progresses the transverse length of the lips is increased, and the muscles of the chin are involved.

The rest of the face muscles, including the zygomatici and the elevator of the upper lip as well as the orbicularis oculi and frontalis, are not affected as a rule. Hence the naso-labial fold is often well marked, and this, coupled with the transverse line of the lips, gives the patient a curious lugubrious expression which is very characteristic.

Paralysis of the soft palate on both sides gradually ensues, the effect of which is to alter the speech very characteristically; and it is important to note that a slight weakness in elevation of both sides will produce much more effect on the voice and on the power of closing the posterior nares than an almost complete paralysis of one-half only. In a paralysis of both sides of the palate, which is sufficient to prevent the patient from closing the posterior nares, the speech is nasal, the difficulty of pronouncing the gutturals *k*, *g* by pressing the tongue against the soft palate is increased; also, as some of the air passes through the nose, the proper amount of pressure to separate the lips is not obtained, so that *p* and *b* become *v* or *f* and *m*.

On trying to inflate the cheeks—taking care that the lips are closed voluntarily or by the finger and thumb—a snorting noise is heard as the air passes through the posterior nares; or, if the anterior nares be closed by the finger and thumb and the cheeks inflated, then, on letting open the anterior nares, the air rushes through and the cheeks collapse.

On phonation, and on a deep inspiration, the soft palate on both sides is very slightly elevated; but the mere hanging of the uvula to one side, or the difference of the height of the two palatine arches, is not of much value, and paralysis of the soft palate can only be said definitely to occur when there is deficient movement on phonation or respiration.

The vocal cords are also liable to be paralysed after the above parts have been involved; and the palsy consists in a difficulty in approximating the cords in phonation and in coughing, so that the voice is low pitched and monotonous, and a proper cough cannot be executed.

In some cases the muscles of mastication—the masseters, temporals, and pterygoids—become involved, and then the patient has difficulty in making the teeth meet, and in masticating the food; and the mouth remains open.

The paralysis and the wasting of the muscles progresses till the patient reaches a most deplorable condition. The features present the characteristic melancholy appearance, the mouth cannot be closed, the lower lip is everted, the tongue lies shrivelled up without any power to move it off the floor of the mouth, the soft palate hangs motionless, or flaps to and fro with respiration. The speech is a slow monotonous mumbling or is lost altogether, the power of phonating a few vowels being all that remains. Mastication is almost impossible, owing in part to the feebleness of the muscles, and in part to the inability of the tongue to keep the food between the teeth.

The act of swallowing is attended by the greatest difficulty and danger. Owing to the paralysis of the tongue the food cannot be pushed backwards by the pressure of this organ from before backwards against the hard palate, but the head has to be thrown backwards to allow the food or liquid to fall backwards by its weight; fluids when they reach the back of the mouth, as the posterior nares are not shut off by the soft palate, readily pass into the nasal cavity and regurgitate through the anterior nares. Again, as the larynx is not drawn up under the tongue, and as the epiglottic muscles and the constrictors of the pharynx are palsied, fluids very easily pass into the glottis instead of into the œsophagus, and frequently produce choking, while the power of expulsion is scarcely assisted by the feeble power of coughing possessed by the patient. Later in the disease the vagus nucleus is sometimes invaded, giving rise to rapid cardiac action and to attacks of dyspnœa.

Owing to the difficulty of swallowing, the saliva accumulates in an extraordinary amount; and, owing to the drooping of the lower lip, the saliva is constantly dripping from the mouth, and the patient saturates many handkerchiefs in the day; on opening the mouth the saliva, which is often thick and viscid, hangs in festoons about the fauces, while every now and again the patient throws back his head in the endeavour to get rid of the excess of saliva by swallowing.

The wasting of the muscles is very marked and does not succeed the loss of power, as in acute nuclear lesions, but goes on step by step with the weakness; it is very gradual and involves fibril after fibril in the tongue. The atrophy of the intrinsic muscles of the tongue can be felt with the finger and thumb, while the mucous membrane is thrown into folds and looks like a half-filled bag. The lips are sometimes not noticeably diminished in size, in others they are decidedly thinner than normal. Wasting of the levator palati and of the vocal cord muscles cannot actually be seen, but their loss of action can be readily ascertained.

The fibrillar contractions are similar to those seen in progressive muscular atrophy; they are best seen in the muscles which are actually wasting, and especially in the tongue, where they give that organ the appearance of a bag half full of worms. The individual fibrils contract one at a time and in succession; movements which are probably due to irritation of the hyperexcitable degenerating nerve-cell in the nucleus supplying the individual fibril.

With regard to the electrical reactions, the affected muscles react to the faradic current as long as there are any muscle-fibres left, which have not degenerated so far as to fail to respond to the current; but as these fibres diminish in numbers as the disease progresses, so the force of the resultant contraction becomes less and less. But these degenerated muscle-fibres which will not react to the faradic current will give the reaction of degeneration with the galvanic constant current; thus we have the muscle giving a slow deliberate contraction with a weaker galvanic current than natural, and responding, on making the current, more readily to the positive than the negative pole. We have, therefore, the following conditions:—in the earliest stages the muscles react to both currents, though not so strongly as normally; later, when about half the fibres have degenerated, the faradic reaction is about half as strong as it should be, while to a fairly strong constant current the quick normal response of the undegenerate muscle-fibres to the negative closure is followed by a slow deliberate contraction of the degenerate muscles; if the minimal current only is tried the slow reaction of the degenerate muscles will be obtained, and probably with the positive rather than with the negative pole. Finally, when all the fibres have wasted there is no reaction to either current.

Reflex action of the larynx and pharynx is almost invariably diminished and after a time abolished; so that on touching the sof palate it is either only slightly elevated or not all, while by tickling th fauces there is great difficulty in producing the reflex actions of retchin and vomiting.

Sensibility is not at all affected, and all forms of sensation are preserved, so that the loss of reflex action is caused on the motor side and not on the sensory side of the reflex arc.

The deep or tendon reflexes in simple uncomplicated bulbar paralysis are not increased, nor are they altered when the disease is associated with progressive muscular atrophy. On the other hand, when the bulbar symptoms form part of the disease called amyotrophic lateral sclerosis, the muscles which close the lower jaw have their so-called tendon reflexes increased in harmony with those of the upper and lower limbs. It is in these cases that the "jaw-jerk" is obtained, and in marked cases "lower-jaw clonus"—a condition which, I believe, I was the first to observe in 1881 and to publish (in *Brain*) in 1886: on placing the finger on the lower teeth and striking it, the lower jaw passes into a clonus similar to ankle clonus.

Though the mental condition of these cases is not affected, the patients are often very emotional, laughing and crying at the same time; and it is extraordinary, considering the perilous and miserable state that they are in, how very good-tempered they are.

The course of the disease progresses from bad to worse, and although in some cases the symptoms may be in abeyance for some months, they increase again and bring the patient to the last stage of the disease.

During the disease the patient is very liable to contract bronchitis or pneumonia, sometimes from particles of food or fluid which may have got into the lungs; and then, owing to the feeble condition of the respiratory muscles, the patient is unable to clear the lungs of mucus, and he succumbs. In his inability to take food the patient undergoes general wasting and weakness, which may end his days.

The disease is prone to extend to the vagus nucleus also and so to paralyse the respiratory movements. Lastly, food may become impacted in the glottis, and, before help can be obtained, the patient is suffocated. The course of the disease is seldom over two years.

Although the above account is the classical description of the disease, anomalous cases have been described by Guinon and Parmentier, and other cases by Rosenthal, Sachs, Bristowe, Seeligmüller, and Eichhorst, where progressive muscular atrophy of the limbs was associated with ophthalmoplegia externa as well as with the usual bulbar symptoms. There seems no reason why the ocular nuclei should not be more frequently associated with progressive muscular atrophy, yet as a matter of experience the combination is very rare.

Two cases, having much importance in the question of age and heredity, have been published by Londe in two brothers, one of whom was paralysed in the facial muscles of both sides, with fibrillar contractions; there was left ptosis, atrophy and fibrillar twitchings of the tongue, salivation, bilateral abductor paralysis of the vocal cords, difficulty in swallowing, and weakness of some of the neck muscles. The other case, in a younger brother of the above, showed definite paresis of frontalis and orbicularis oculi of the facial muscles, followed by commencing weakness of the lower face muscles. Londe considered that the above cases were examples of a true family progressive bulbar paralysis occurring in childhood, the chief characteristics being that the upper facial muscles were attacked, and, especially, that the disease began in those muscles.

It cannot be certainly stated that these cases were due to a lesion of the medulla, as no autopsy was recorded.

Pathology.—The disease is essentially a slow degeneration of the cells of the motor nuclei of the bulb. The changes which cause the muscular wasting are found in the nuclei of the hypoglossal nerve of both sides, the vagus, and its accessory nucleus.

The first accounts of definite post-mortem changes were published by Charcot and Joffroy, who found chiefly atrophy of the cells in the hypoglossal nucleus, while the combined nuclei of the glosso-pharyngeal, vagus, and its accessory nerve were but slightly affected.

It should be mentioned that, according to Drs. Tooth and Turner, no lesion in the 9th, 10th, and 11th nuclei could be found in a case of bulbar paralysis examined by them, though the hypoglossal nucleus was much affected, and there was paralysis of the soft palate and vocal cords. According to these authors, only certain of the motor cranial nerves were affected; beginning from above, the motor nucleus of the 5th and the nucleus of the 7th (facial) had their cells atrophied and granular at the highest part of the nucleus, but lower down the cells were more normal. The ascending loop of the 7th nerve, which lies dorsally to the posterior longitudinal bundle, was completely degenerated, the nerve-fibres being absent. The issuing 7th nerve-trunk was not, however, completely atrophied, but contained many healthy nerve-fibres; thus showing that some of the fibres proceeded from healthy nuclei. The 9th, 10th, and 11th nuclei were healthy; but in the 12th nucleus the cells were much atrophied, and also the fine network of fibres in the nucleus. The small-celled nucleus of the hypoglossal (Roller) escaped.

The cells of the nuclei gradually waste and lose their processes, and are at length represented by small angular bodies. The products of the degeneration of the cells, in the form of granule corpuscles, are met with; and in some cases interstitial changes in the connective tissue with enlargement of the blood-vessels have been observed.

In uncomplicated cases, and in those associated with progressive muscular atrophy, there are no changes in the pyramidal tracts; it is only in cases of amyotrophic lateral sclerosis that sclerosis of the pyramidal tracts is also found.

Of the bulbar nuclei the hypoglossal nucleus suffers most frequently; then the accessory to the vagus, the vagus, and less frequently the glosso-pharyngeal nucleus, the motor nucleus of the 5th nerve, and the facial nucleus itself.

The nerve-trunks which supply the affected muscles are gray in colour and diminished in size; and in some nerves like the hypoglossal very few healthy fibres can be found, while the interstitial connective tissue is increased.

The muscles themselves have their fibres much atrophied. In some cases the individual fibres are smaller, or show signs of granular or fatty degeneration. The changes are precisely the same as those found in progressive muscular atrophy.

The relation of the grouping of the affected muscles to the nuclei involved is of great importance. It has been noticed for some time that in these cases the facial muscles do not waste as a whole; but that, whereas the orbicularis oris was associated with wasting of the tongue and paralysis of the palate and laryngeal muscles, the middle face muscles, the elevators of the angle of the mouth were much less frequently affected, and the orbicularis oculi and frontalis practically never; so that there must be some association between the tongue muscles, the orbicularis oris, the soft palate, and the laryngeal muscles. In the remarks on the anatomy and physiology in Dr. Turner's article, attention has been called to the arrangement which limits the function of the facial nucleus to so few of the muscles of the face—only the elevators and depressors of the angle of the mouth; and with this observation must be taken a case, observed by Sir William Gowers, of poliomyelitis due to nuclear paralysis, where the orbicularis oris escaped entirely, though the rest of the face was absolutely paralysed, as showing that the orbicularis oris is probably not supplied by the facial nucleus. There seems no reason to doubt that the reason of the tongue and orbicularis oris being affected together is that both are supplied by the hypoglossal nucleus, while the close association of the soft palate and the vocal cords would be due either to the implication of the accessory nucleus, which is close to the hypoglossal, or to the motor supply of muscles of these parts from the hypoglossal nucleus itself through the accessory nerve, as suggested by Tooth and Turner.

We have no means of ascertaining the cause of the degeneration of the nerve-cells, and it is quite possible that the changes may be due to toxic influences.

Prognosis.—This is always very bad. The duration of the disease is seldom more than two years, and usually ends the life of the patient. The prognosis is especially unfavourable in those cases which are associated with progressive spinal muscular atrophy.

Treatment.—It seems impossible to arrest the disease, but it is well to try to do so if only for a time, and meanwhile to make the existence of the patient as comfortable as possible.

Patients should be warmly clad, and be careful to avoid the risks of bronchitis or pneumonia.

Feeding is always a difficulty, especially in the later stages, but it is very important to keep up the strength of the patient by a sufficient quantity of food. Semi-solid pultaceous foods are swallowed better than liquids or solids. Meat and fish should be pounded, passed through a sieve, and made up into a thick consistency with soups. Meat jellies can also be managed. All hard substances, such as the crust of bread, should be carefully avoided. When swallowing is impossible, artificial feeding by a funnel and a soft tube passed through the mouth or nose must be used. While they are able to swallow, these patients should never be left alone while eating, as the sudden impaction of food in the glottis may cause death before an alarm can be given.

With regard to internal treatment, quinine, arsenic, iron, and strychnine should be given. The last may advantageously be given in the form of hypodermic injection, as recommended by Sir William Gowers who has seen improvement take place under it, of gr. $\frac{1}{60}$ of the nitrate of strychnine with gr. $\frac{1}{36}$ to $\frac{1}{24}$ of morphine.

Electrical treatment has frequently been applied, but I cannot say that I have seen much good from it ; if it be employed, the constant current should be used, not the faradic, and the negative pole should be applied direct to the muscles affected, the positive pole being over the back of the neck. Galvanisation of the sympathetic has also been employed.

Of the other forms of chronic disease affecting the nuclei of the bulb, tumours may be mentioned. These very rarely affect the nuclei of the 9th, 10th, 11th, and 12th nerves ; but tumours such as gliomata or tuberculous deposits may occur on one side within the medulla at its junction with the pons, and cause complete paralysis of the facial nerve on that side, conjugate deviation of the eyes to the opposite side, and paralysis of the opposite arm and leg. These symptoms are produced when the 6th nucleus, the 7th nerve, and the pyramidal tract of one side are involved.

Infra-nuclear paralysis.—The symptoms of these lesions differ according as the position of the lesion is within the medulla itself, or outside the medulla pressing on this structure, or implicating its nerves before they emerge from the cranial cavity. The symptoms will have to be carefully distinguished from those produced by lesions of the bulbar nerves after they have left the cranial cavity, which hardly come under the title of bulbar paralysis.

The infra-nuclear lesions within the medulla must of necessity be very limited in range, as they take effect on the bulbar nerves only in their passage between their nuclei at the posterior part of the medulla and their exit from the surface of the medulla. The acute form has already been referred to (p. 225), and the most likely lesion would be softening caused by thrombosis of one of the vessels entering the medulla from the vertebral artery, and the symptoms would be sudden paralysis of the tongue muscles of one side, or of the soft palate and vocal cord of one side, with loss of power and perhaps anæsthesia of the opposite limbs and half of the body with escape of the face, while the trapezius and sternomastoid muscles supplied by the spinal accessory would not be affected. The half of the tongue and the soft palate would waste, and give the reaction of degeneration to electrical testing. A tumour within the medulla at this part would also give the same symptoms with a gradual onset, but the condition is very rare.

The *extra-medullary lesions* are more common and definite than the intra-medullary.

The symptoms may be due to a tumour pressing on the nerves and on the medulla, or to meningitis, which may be simple or syphilitic. Of these, syphilitic gumma and syphilitic chronic meningitis are the most common causes.

In syphilitic meningitis the membranes are very much thickened and compress the nerves passing through them, a condition which may be associated with a gumma in the neighbourhood or be independent of it.

The symptoms consist in a gradual paralysis of one-half of the tongue, one-half of the soft palate, and one vocal cord, all on the same side. This combination was first pointed out by Dr. Hughlings Jackson (14) in 1864, in cases of lesion outside the medulla, and it is due to the implication of the accessory to the vagus and the hypoglossal nerves. In some cases the spinal accessory is also affected, whereby the sterno-mastoid and trapezius of the same are paralysed; and also, although I have not yet had the opportunity of observing a case, I anticipate from the experiments which I made with Mr. Horsley (2) that the depressors of the hyoid bone, which are supplied by the 1st and 2nd cervical roots, and not by the hypoglossal nerve, would be paralysed on the same side. These two last symptoms are of very great importance, as paralysis of the tongue, soft palate, and vocal cord on one side might be caused by an intra-medullary lesion affecting the hypoglossal nerve and the accessory to the vagus; but such a lesion, unless it extended down the interior of the medulla and cervical cord, would not cause paralysis of the depressors of the hyoid and of the trapezius and sterno-mastoid, and if it were so extensive, it would probably cause paralysis of all four limbs, by involving the decussation of the pyramids.

The affected muscles waste, and, being cut off from their trophic centres, the bulbar nuclei, give the electrical reaction of degeneration.

The symptoms do not cause the difficulty of the other forms of bulbar paralyses, as they are unilateral and the patient is able to speak, swallow, and cough by means of the opposite unaffected side.

The *prognosis* of the infra-nuclear paralyses varies very much according to their cause and their situation within or without the medulla. Intra-medullary lesions are more serious than extra-medullary, but tumours otherwise than syphilitic must soon be fatal. The least dangerous condition would be syphilitic chronic meningitis and gummata, which are often amenable to treatment, though by no means invariably.

Treatment consists in antisyphilitic remedies, even in the absence of any history of syphilis; and for children in cod-liver oil and syrup of iodide of iron, on the chance of the growth being tuberculous.

If an extra-medullary tumour could be diagnosed with any certainty, it might be possible to reach it through the posterior fossa of the skull and to remove it surgically, though I am not aware that such an attempt has yet been made.

Extra-cranial causes.—Although they are not included under the name "bulbar paralyses," we must remember that lesions of the lower cranial nerves outside the cranium give rise to symptoms which are often very difficult to diagnose from those of intra-cranial bulbar lesions. These lesions may be enumerated as *caries of the cervical vertebræ, cellulitis of the neck, tumours, and multiple neuritis.*

As an example of an extra-cranial tumour, I recently had a case of complete paralysis of both sides of the soft palate in which reflex action and faradic excitability were lost, but the galvanic reaction of degeneration was present. The patient had no paralysis of the tongue or of the vocal cords, but the whole of the right 5th nerve, motor and sensory, was involved, with complete loss of taste for that side of the tongue. At the autopsy a sarcomatous tumour was found on each side at the back of the pharynx, but not extending across the middle line; and it had also spread backwards into the cranial cavity through the foramen lacerum medium on the right side, and attacked the 5th nerve near its root. The paralysis of the soft palate was considered during life to be due to inclusion of the pharyngeal plexus in the growth, while the freedom of the tongue and vocal cords was against the disease being intra-cranial, so far as the bulb was concerned.

Neuritis may affect single nerves like the hypoglossal, and I have seen one case of hemiatrophy of the tongue in diabetes.

Very rarely cases of multiple neuritis occur in this region; I have recently had a case in a young girl, who suffered from severe pains in the arms and legs followed by anæsthesia, loss of power, and wasting in the muscles of the hands and feet and loss of faradic excitability; associated with these symptoms were complete loss of power in both sides of the face, including the frontalis and orbicularis oculi, weakness of the levator palati, especially on the right side, with inability to close the posterior nares, weakness and atrophy of the right half of the tongue, difficulty in approximating the vocal cords, and paralysis of the diaphragm. Though I had not the opportunity of verifying the diagnosis, I think from the associated symptoms in the limbs that the bulbar symptoms were due to an acute peripheral neuritis. Two somewhat similar cases have been described by Dr. Buzzard, due probably to syphilis, and both these patients recovered.

Bulbar paralysis without apparent anatomical changes.—From time to time cases of paralysis, apparently bulbar, have been recorded without the discovery of any pathological change after exhaustive necropsies.

A case was described by Sir Samuel Wilks, in 1873, in a girl with general weakness followed by symptoms of bulbar paralysis, indistinct speech, difficulty in coughing, but without paralysis of the limbs; the respiration became affected, and she died in a few hours. Microscopical examination revealed nothing pathological in the medulla.

A case was also published by Dr. L. E. Shaw of general weakness of the whole body, with difficulty of speaking and swallowing, in which nothing abnormal was discovered in the medulla; as there was no local wasting, and there was increase of the deep reflexes, the case would seem to resemble the form of amyotrophic lateral sclerosis where the nuclei are not affected; but no mention was made in the microscopical report of sclerosis of the lateral columns or pyramidal tracts.

Another case has been described by Oppenheim in which all the symptoms of chronic bulbar paralysis occurred; yet nothing was discovered, although the nuclei of the bulbar nerves, their roots, and also the peripheral nerves were carefully examined.

Four cases have been published by Dr. Pineles of bulbar paralysis in which there was paresis of the eye muscles, difficulty of swallowing and of articulation, with weakness of the limbs and a marked feeling of lassitude; death occurred suddenly, and no changes were found in the medulla.

Another similar case has occurred in which Meyer, in Marchi-stained preparations, found degeneration of the intra-medullary spinal roots; so that it is possible that in these cases there is some latent pathological change.

Another class of cases of bulbar paralysis without anatomical changes are those described by Strümpell, which he calls asthenic. Here the patients are young people—one a girl aged twenty—who readily feel tired, and have slight pains about them, then ptosis, difficulty in speaking, swallowing, and biting; later the muscles of the neck, back, and limbs are attacked, and lastly the respiratory muscles. The sphincter ani and sphincter vesicæ are not affected. There is no atrophy, the knee-jerks are slightly diminished, but there is no change in the electrical reactions. Remissions usually occur, but in the above case the patient died suddenly, after several attacks of dyspnœa, ten months after the onset. At the autopsy nothing pathological was found macroscopically or microscopically, and it is possible that the condition may be due to some organic poison.

The general diagnosis of bulbar paralyses is one of the greatest importance, and often also one of great difficulty.

The first point is to discriminate between the supra-nuclear lesions on the one hand and the nuclear with the infra-nuclear on the other.

In the supra-nuclear lesions the muscles do not waste, except from disuse, there are no fibrillar contractions, the electrical reactions are normal, reflex actions are preserved, and in some cases the emotional movements of expression; so that although the patient's face may be absolutely motionless to volitional efforts and he cannot cough voluntarily, he can cough, smile, and yawn reflexly, while the reflex actions of the soft palate and pharynx are also preserved.

Whether the emotional actions are preserved in lesions of the middle of the pons has not yet, I believe, been ascertained.

On the other hand, in all nuclear and infra-nuclear lesions affecting the bulbar nerves, the muscles waste and give the electrical reactions of degeneration, and they sometimes present fibrillar contractions, while all reflex actions are either diminished or abolished altogether.

In supra-nuclear paralyses the pseudo-bulbar paralyses, due to lesions of the cortex or internal capsules, are characterised by a history of two attacks of hemiplegia involving the face, tongue, perhaps the limbs, first on one side and then on the other, and the symptoms occur after the second attack; whereas in lesions just above the nuclei the symptoms may come on after one attack, and the limbs may not be affected.

In the nuclear and infra-nuclear cases the first distinction is into acute and chronic cases. Taking the acute cases first, we have to separate them into those of sudden and those of rapid onset, the former being due to occlusion of a vessel or to a small hæmorrhage, while the latter, being due to an inflammatory condition of the nuclear cells, would be attended by fever and vomiting.

The diagnosis of acute nuclear from acute infra-nuclear lesions within the medulla would depend on the distribution of the symptoms. In nuclear paralyses it would be very difficult (except in the inflammatory form) for one hypoglossal nucleus to be affected without the other, and, furthermore, the orbicularis oris would probably be affected while the limbs would escape; whereas a lesion of the hypoglossal nerve in its intra-medullary course would not affect the orbicularis oris, and probably would cause weakness or anæsthesia of the opposite limbs.

The extra-medullary lesions are usually chronic with a gradual onset, and are nearly always unilateral; while the association of paralysis of the sterno-mastoid, trapezius, and also of the depressors of the hyoid bone, on the same side as the paralysis of half the tongue, the soft palate, and one vocal cord, would make the extra-medullary position of the lesion almost certain.

The progressive bilateral character of chronic bulbar paralysis should prevent any difficulty in its diagnosis.

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P.S.—With the cases related on p. 236, I may bring forward the following instance of bulbar disease, the issue of which was a great shock to me and a painful lesson. Miss ——, aged 18, of good personal and family history, "caught cold six months previously, followed by stiffness of the tongue and jaws"; this stiffness remained for a few weeks and then disappeared. She now moves her tongue and jaws, and indeed every muscle of the face, throat, and orbit, quite normally. Palatal reflexes normal. Jaw reflex not tested. But of late she has begun to talk oddly, "as if with a potato in her mouth." This gets worse as she proceeds, when her utterance becomes involved, and she stops. Phonation does not fail but articulation, as if there were an incoördination of the muscles of speech. I put her to read from a book; she began well, but soon the disorder set in, and she became unintelligible. Her friends told me she read much better when at home quietly, "not nearly so badly as before me." A few weeks ago a difficulty in swallowing came on, "she choked over her meals, more when she was tired, that is, at the later meals of the day." The account of it seemed quite like that of "neurotic spasm of the œsophagus." She is, however, a healthy, nice-looking girl, and I find no definite evidence of hysteria or other neurosis, or of palsy or atrophy. Visual field normal. I talked the case over with her own medical man; we could find no other evidence of organic disease, except, by the way, that the forefinger of the left hand seemed weak and fumbling, and she said it was "numb," so that she had had to give up needlework.

We made a provisional diagnosis of hysteria, yet with reserve, as the disorder of speech seemed a strange one. However, on some moral discipline and tonic medicine with valerian, she improved so much that our apprehensions were lulled to rest, and I did not see her again. We had talked of sending her to the Queen's Square Hospital, but now she seemed in the way of recovery. One evening, three weeks after her call on me, her symptoms got worse again, especially the swallowing, and her mother, seeing this, warned her somewhat sharply to control herself.

The next day the patient came into her mother's bedroom about 8.30 A.M.—to protest, as well she might, poor child, that she really could not help these eccentricities—when suddenly she fell to the ground, was convulsed, turned blue, and died at her mother's feet. She could scarcely have died by choking with food as this terrible event happened before her breakfast. To suggest a necropsy under such circumstances seemed to her medical man impossible, and a certificate of death in an epileptic fit was given. I was at a loss to comprehend how this patient could have progressive structural disease of the medullary centres without producing palsy or wasting of the muscles concerned, but the case was probably one of those anomalous fatal instances in which the medulla has been found apparently free from static disease. In this opinion Dr. Beevor coincides, while pointing out that the usual mode of death is by dyspnœa.

ED.

DISEASES OF THE BRAIN

THE EXPERIMENTAL PATHOLOGY OF THE CEREBRAL CIRCULATION

The cerebral pulsation.—As early as 1750, knowledge of the respiratory nature of the oscillations of the brain volume was established by Schlichting.

In the last thirty years the movements of the brain have been accurately recorded by Salathé, François Franck, Mosso, Elder, Hutchinson, and many others. Records have been taken from men with cranial defects, and from infants by way of the fontanelle.[1] It has been asserted that the brain cannot pulsate in the closed cranium, on the ground that the brain fills the rigid, unyielding box of the skull in which it lies enclosed. The inaccuracy of such a statement can easily be shown by the following means:—After the skull of a dog has been trephined and the dura opened, a piece of metal tubing, on the lower end of which a thin rubber membrane is tied, is screwed into the trepan hole. This membrane, after the introduction of the tube, lies in apposition with the brain. To the other end of the metal tube is fastened a piece of glass tubing of fine bore. This glass tube is filled with water containing a bubble of air, to act as an index. In its turn it is attached, by a T-tube, to a pressure bottle and a mercury manometer. Now, in using this simple apparatus, the position of the air-bubble index is marked on the glass tube when the apparatus is held in the horizontal position, and the pressure is nil. The gauge is then screwed into the skull, and the air index, which at first is forced out, is brought back to its original position by raising the pressure bottle; that is to say, the brain, which is slightly bulged outwards into the trephine hole, is forced into that flattened position which it naturally must assume against the closed cranial wall. The pressure of the cranial contents against the skull wall is exactly balanced, and the skull becomes once more a closed cavity, and on this procedure the elevation of the manometer gives the normal intracranial tension. The air index will then exhibit cardiac and respiratory undulations of intracranial pressure.

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[1] The tracings show cardiac and respiratory oscillations. The pulse curve is usually anacrotic; that is to say, a small wave marks the ascending limb of the curve. This anacrotic wave is probably produced by reflection of the primary wave from the wall of the cranium.

The diastole and systole of the brain are made possible in two ways:—

1. By the ebb and flow of the cerebro-spinal fluid. The occipito-atlantal and other vertebral ligaments may extend in cerebral diastole, and allow the fluid to escape from the cranial cavity. The pulsation of the fluid can easily be observed by trephining the lamina of the atlas, screwing a tube therein, and connecting this tube with a water manometer. This mechanism is of little importance. Elder has shown that when a small quantity of Prussian blue is injected into the subarachnoidal space, and the animal is killed a short time afterwards, the blue matter is found not to have shifted its position. Thus there cannot be any marked translation of cerebro-spinal fluid synchronous with the cerebral movements.

2. By expression of the cerebral veins. In the dog it is a simple operation to screw a tube into the torcular Herophili. When this tube is connected with a water manometer the cerebral venous pressure can be recorded. Such a venous manometer exhibits all the cardiac and respiratory oscillations; the latter in a very marked way.

The brain is an organ which, like any other organ of the body, pulsates with each stroke of the heart; but, owing to its peculiar condition enclosed as it is in an unyielding box, the cardiac pulse is not entirely spent in distending the arterioles and capillaries, but is transmitted to the venous sinuses. The brain, expanded by the stroke of the cerebral arteries, compresses the cerebral veins against the cranial wall. The respiratory oscillations are caused by the variations of pressure in the right side of the heart. In expiration the blood is dammed back into the sinuses; in inspiration the veins are emptied by the suction of the thorax.

The brain, unlike such an organ as the kidney, expands more during expiration than during inspiration. This is due to the absence of efficient valves in the cranial and vertebral veins, and to the immediate continuity of these veins with the right auricle and with the venæ cavæ. If the torcular Herophili be opened in the freshly-killed animal, and the abdominal and thoracic veins be compressed, venous blood can be driven out of the torcular in a continuous stream. Likewise, in the human subject, fluid can be driven from the femoral vein out of the cerebral sinuses.

Pathologically the movements of the brain can become increased or diminished. Any cause that produces increased tension of the dura may, when the skull is trephined, stop the apparent pulsation of this membrane. As the pulsation of the brain takes place always in the direction of least resistance, the brain pulsation is but slightly marked in conditions of normal pressure; that is to say, when the heart and the respiration run smoothly. If the skull be trepanned during the condition of acute cerebral compression, the pulsation may be visibly increased owing to the slow, powerful beats of the heart, and the stertorous breathing.

The pulsation may be imperceptible when the brain is bound down by adhesions in the neighbourhood of the trephine hole; and, again, whenever the cardiac systole is feeble and the respiration shallow.

It is to be concluded, then, that—(i.) The pulsations of the brain exist in the closed cranium. (ii.) These pulsations are of cardiac and respiratory origin. (iii.) The cardiac pulse is transmitted from the cerebral arteries to the cerebral venous sinuses. (iv.) The brain stands in closer relation to the vena cava than to the aortic pressure. Thus, normally, its greatest expansion is in expiration, and not in inspiration. If, however, the respiratory oscillations of arterial pressure become greatly magnified this condition may be reversed. (v.) The cardiac systolic expansion of the brain within the closed cranium is rendered possible by the ebb of the cerebro-spinal fluid into the less rigid vertebral canal, and by the expulsion of the blood from the cerebral veins and sinuses. (vi.) Increased tension of the dura mater decreases its exhibition of the cerebral pulse. (vii.) On opening the dura, provided the brain movement be unrestricted by local adhesions, the extent of the pulsation will depend on the magnitude of the cardiac oscillations of arterial pressure, and on the depth of the respiratory movements.

The cerebro-spinal fluid.—Neither the brain nor the spinal cord possesses true lymphatic vessels; the lymph finds its way out of these organs by means of perivascular spaces in the tunica adventitia of the blood-vessels; these perivascular spaces communicate on the one hand with the cell spaces, on the other hand with the subarachnoidal space.

The central canal of the cord and the ventricles of the brain are connected with the subarachnoidal space by the foramen of Majendie, and two other foramina in the lateral recesses of the fourth ventricle, by the side of the flocculus cerebelli. The subarachnoidal space is continuous, extending thoughout the central nervous system; anatomically, it is separated from the subdural space. The subarachnoidal space is in continuity with the lymph spaces in the optic, auditory, and other nerves. The anatomical lack of continuity between the subarachnoidal and the subdural spaces is not physiologically important; for it is only by very gradual and most careful injections of coloured gelatine that the continuity of the true cerebral with the spinal subarachnoidal space can be shown. On opening the dura in the living animal, with the utmost care, the fluid within the cranium, if any, is seen to be in the subdural space. Saline or serum injected subdurally into the cranium at a pressure just above intracranial pressure runs with ease into the subarachnoidal space of the vertebral column.

The plates, copied from Key and Retzius' great monograph into most of the anatomical works of to-day, give an entirely erroneous idea of the contents of the cranium in the living animal. Therein we find the arachnoidal cisterns at the base of the brain distended with an artificial injection, and the tentorium cerebelli pushed upwards and out of place.

In the living animal the chief content, besides the brain substance, is blood, not cerebro-spinal fluid. The living normal brain, with its circulating blood, almost entirely fills the cranium, and the fluid that moistens its surfaces is little more in amount than the synovial fluid in a joint.

The cerebral subarachnoidal space is a potential rather than an actual space, except in those few places where inequalities of the brain surface are rounded off by small collections of fluid beneath this membrane. In the vertebral canal the fluid is somewhat more in amount; and probably, by its presence there, it wards off compression from the spinal cord during the flexions and torsions of the vertebral column.

The hydrostatic pressure of the cerebro-spinal column of fluid during changes of posture has been supposed to be of importance. This is not so, for the pressure in the blood-vessels under the influence of gravity varies in the same sense as the pressure of the cerebro-spinal fluid. Moreover, the fluid leaks into the veins at any pressure above venous pressure. Thus in the erect posture the lumbar portion of the cord cannot be rendered anæmic by the hydrostatic pressure of the column of cerebro-spinal fluid.

Experiments on the behaviour of fluids injected into the cranio-vertebral cavity have led to results of very great interest. On trephining the lamina of a vertebra and screwing in a tube, normal saline can be driven into the vertebral canal. If a second trephine hole be made in the parietal region none of the injection into the vertebral canal can be driven out from this hole; the mid-brain is floated up by the fluid into the isthmus of the tentorium cerebelli and plugs this up, while the great brain likewise moves upwards and plugs the trephine hole in the parietal region. If, on the other hand, the saline be injected into the parietal hole, it can, in most cases, be driven through the cranio-vertebral canal and out of the hole in the vertebral column. The whole central nervous system can thus be irrigated freely. In some cases, when the pressure is low, and nearly always, if the pressure of the injecting fluid be high and applied suddenly, this cannot be done. By the pressure of the fluid the cerebrum is driven into the isthmus of the tentorium cerebelli, and blocks this up so that no fluid can be expelled. If the cerebrum be removed from the cranium of a dog, and the empty cerebral chamber be filled with water, none of the fluid leaks through the foramen magnum. This shows how completely the cerebellar chamber is filled by the brain mass within it.

Saline or serum injected at any pressure above the cerebral venous pressure is found to disappear from the cranio-vertebral cavity; the higher the pressure the more rapid its disappearance. If the saline be coloured with methylene blue, the fluid can be traced passing straight into the cerebral veins and venous sinuses. In so short a time as five to ten minutes the blue colour may be found excreted into the stomach or bladder. In this time the lymphatics of the neck are not coloured, and not until the lapse of an hour's steady injection do the deep cervical lymphatic glands begin to be tinged with colour. It is clear that the rapid absorption of fluid from the cranio-vertebral cavity takes place by means of the veins and not by way of the lymphatics. This statement holds equally true for the pleural and peritoneal cavities. Fluid will not remain for any length of time in the cranio-vertebral cavity at a pressure above the cerebral venous pressure. This is a fact of the utmost importance in the pathology of cerebral compression.

In the normal healthy state the amount of cerebro-spinal fluid is very slight. On opening the occipito-atlantal membrane in the dog but a few cubic centimetres at the most will be obtained. By continuously tapping off the fluid Falkenheim and Naunyn obtained from dogs 36 to 240 cubic centimetres in twenty-four hours, but as soon as the cranio-vertebral cavity is opened the conditions are no longer normal. After such opening, the rate of transudation of the fluid seems to depend directly on the cerebral capillary pressure. The cerebro-spinal fluid is normally maintained at the pressure in the cerebral veins. If the fluid be allowed to escape from the cranio-vertebral cavity, the pressure within this cavity then falls to zero, while the cerebral capillary pressure remains unaltered. Consequently transudation from the capillaries becomes continuous, hence the large amount of fluid obtained in some cases of fracture of the skull. The fluid escapes just as any raw surface weeps, as, for example, after an abrasion of the skin. Since high arterial pressure increases transudation, and high venous pressure prevents absorption, these are the most favourable conditions for increasing the outflow of cerebro-spinal fluid from a cranial opening.

The cerebro-spinal fluid should be regarded as the lymph of the brain, its secretion and resorption being explained on the same lines as other lymphatic exudations. The difficulty of obtaining a sufficient quantity of normal cerebro-spinal fluid from animals has prevented adequate study of this secretion, and the whole question of lymph secretion is at present in too unsettled a state to be discussed with much profit.

Professor Halliburton and others have found that cerebro-spinal fluid, drawn fresh from the subarachnoidal space, possesses a peculiar chemical composition. The presence of a reducing body, belonging to the pyrocatechin class of compounds, is the most characteristic feature. Cerebro-spinal fluid contains but traces of serum albumin, the proteids present being in the form of albumoses. The fluid does not clot. The specific gravity ranges from 1005 to 1010. The inorganic salts = about 8 per 1000; the organic matter = about 1 to 5 per 1000. The fluid does not coagulate on boiling, it gives a precipitate with cold nitric acid, soluble on heating, a pink reaction with a drop of copper sulphate and caustic potash, and reduction of Fehling's solution.

If the fluid be drawn off again and again it changes in character, and more nearly approximates to the composition of serum. The same change follows the repeated tappings of the sacs of spina bifida and hydrocephalus.

In a case which I was permitted to examine, by the kindness of Dr. St. Clair Thomson, cerebro-spinal fluid continually dripped from the nose. In all probability the fluid escaped from some perforation in the plate of the ethmoid bone. This fluid, according to the analysis of Professor Halliburton, possessed the characteristic properties of cerebro-spinal fluid. When the patient was made to strain, as at stool, the rate of flow of the fluid was doubled. On qualitative analysis it was found by Halliburton that the percentage of total solids in the fluid passed during straining was less than half that passed when the patient remained passive. The same result followed compression of the patient's abdomen. This case confirms the conclusion, arrived at previously, that the secretion, so soon as the cranium is opened, becomes an exudation which depends on the pressure within the cerebral capillaries. Either the act of straining, or passive compression of the abdomen, markedly raises the general venous, and thus the cerebral capillary pressure. From the above considerations it may be concluded that:—

(i.) The brain, with its circulating blood, almost entirely fills the cranial cavity in the living animal. (ii.) Fluid escapes directly into the cerebral veins from the subarachnoidal or subdural spaces at any pressures above cerebral venous pressure. (iii.) So long as there is cerebro-spinal fluid within the meningeal spaces, the pressure of this fluid cannot be other than that of the cerebral veins. (iv.) The cranial vertebral cavity can be irrigated with normal saline at a low pressure. This can be effected through one trephine hole without any counter-opening, and is a perfectly harmless operation. (v.) No local pathological increase of cerebral pressure can be transmitted by the cerebro-spinal fluid to distant parts, because this fluid can never be retained in the meningeal spaces at a pressure higher than that of the cerebral veins. (vi.) It is thus probable that hydrocephalic distension of the ventricles of the brain may be relieved by establishing a drain into the subdural space. (vii.) The cerebro-spinal fluid preserves its peculiar constitution under normal conditions of pressure only. If it be drawn off, its place is taken by a serous exudation. The rate of transudation when the cranio-vertebral cavity is opened depends directly on the difference between cerebral capillary and atmospheric pressure.

The principles of the cerebral circulation.—The quantity of blood within the cranium is almost invariable, "for being enclosed in a case of bone, the blood must be continually flowing out of the veins that room may be given to the blood which is entering by the arteries" (Monro). "In such a cavity as the cranium the blood cannot be diminished below a certain quantity, unless something entered in to supply its place, and, in the language of the old philosopher, prevent a vacuum" (Abercrombie).

The only substance which can enter into or forsake the craniun, when the blood quantum varies, is the cerebro-spinal fluid. Now this fluid is of insignificant amount. "It is well known that there is found very little of the cerebro-spinal fluid—in fact, in general little more than what is sufficient to moisten the surface of the membranes—in the interior of the cranium in healthy persons up to the middle of life. Under these circumstances the quantity of cerebro-spinal fluid that could be displaced in the interior of the cranium must be trifling."

The condition under which the cerebro-spinal fluid is increased in amount is atrophy of the brain substance. A long continuance of low arterial pressure when the head is elevated above the body may produce the same result, for the pressure of the blood in the cerebral vessels under the influence of gravity may become negative, and the circulation through the brain fail. As the capillary walls become damaged, serous fluid will then filter through, owing to the elasticity of the blood-vessels; for these strive to return to the position of equilibrium. Thus the vessels collapse and the serous fluid increases. In some men who died of cold in a snowstorm Kellie found more than four ounces of serous fluid in the ventricles of the base of the brain.

The examination of the condition of the cerebral vessels after death is a very deceptive method of obtaining information as to the amount of blood in the brain prior to death. After death conditions alter: serous fluid or gas may pass out of the vessels into the cranium; by alteration in the position of the body, or pressure on the abdomen and thorax, the cerebral vessels can be injected with venous blood, the arteries emptied, and the cerebro-spinal fluid expelled. For example, the ordinary conditions of the circulatory system which are found after death from asphyxia can be entirely removed by the employment of vigorous artificial respiration on the dead body. These are facts too often neglected.

The truth of the Monro-Kellie doctrine—that the blood quantum of the brain does not vary—is easily verified. If the skull be trephined, and the dura opened, a glass window can be screwed into the hole. On then placing the animal in the vertical feet-down position, and inhibiting the heart, the brain does not collapse away from the window, although the arterial pressure falls to zero. On the other hand, if the glass window be faultily placed and allow leakage into the cranial cavity, air will pass within, the blood be expressed, and the brain collapse under atmospheric pressure.

While the cranium is closed, large alterations of blood volume are impossible; in the open cranium the atmospheric pressure is brought to bear upon the cerebral capillaries, and the physical conditions of the cerebral circulation are altered. The cerebral capillaries are emptied of blood whenever air freely enters the cranial cavity, and the arterial pressure falls below that which is required to elevate the blood from the heart to the brain, and to overcome the resistances due to the viscosity of the blood in the arteries.

Although the circulation in the brain has been frequently investigated, many fallacious methods have been employed. Accurate results can be obtained by simultaneously recording the aortic pressure, the pressure in the superior vena cava, the intracranial pressure, and the cerebral venous pressure, the cranium being, as in the normal condition, a closed cavity. As a control to elucidate certain points, the pressure of the cerebro-spinal fluid must also be recorded.

The required observations can be carried out thus. The aortic and vena cava pressures are obtained by passing canulæ down the carotid artery and jugular vein respectively. These canulæ are connected with recording manometers. The intracranial pressure is observed by means of the brain pressure gauge. The cerebral venous pressure can be recorded by screwing a tube into the torcular Herophili, and connecting this with a manometer. Lastly, the pressure of the cerebro-spinal fluid can be measured by trephining the lamina of the atlas, screwing a tube into the trephine hole after the dura has been opened, and connecting this with a manometer.

By this method of research it has been determined that the cerebral venous and intracranial pressures passively follow the slightest changes in aortic or vena cava pressures. No evidence has been forthcoming of any independent change pointing to the existence of any vaso-motor nerves supplying the brain. No positive proof of vaso-motor changes in the brain has appeared on stimulation of the vaso-motor centre, the central end of the spinal cord after division of the cord in the upper dorsal region, or on stimulation of the stellate ganglion; that is to say, after the whole sympathetic supply to the carotid and vertebral arteries has been excited.

Conclusive evidence is not forthcoming of the existence of any local vaso-motor mechanism, such as might be called into play by the injection of drugs into the cerebral circulation. Such researches as have yielded evidence of cerebral vaso-motor nerves are rendered worthless by the omission of records of vena cava pressure.

In every experimental condition the cerebral circulation passively follows the changes in the general aortic and vena cava pressures, so that even if the existence of cerebral vaso-motor nerves be postulated, their power must be insignificant.

In a recent research* Dr. Gulland has obtained *anatomical* evidence of the existence of nerve-fibres ramifying over the pial vessels. These may be vaso-motor in function. The primary function of the muscular wall of the blood-vessels is to maintain tone; the exciting cause of the tonic condition is probably the pressure or quality of the blood. The vaso-motor nerves increase or inhibit the excitability of the muscular wall, and thus vary the vascular tone, but the tone soon returns and persists when these nerves are severed. In the splanchnic area the vaso-motor influence is greatly developed; in the locomotor organs but slightly. During asphyxia the limbs are passively dilated by the effect of splanchnic constriction, nevertheless the arteries in the limbs are no less muscular than those in the mesentery. In the pulmonary circulation active changes are easily masked by passive effects brought about by changes in the vena cava and aortic pressures. In the cerebral circulation this may be still more strikingly the case. Trustworthy experimental methods have as yet failed to demonstrate any action of vaso-motor nerves on the cerebral vessels.

When in any part of the body the arteries contract, the blood volume in the area diminishes, the veins empty, the whole organ shrinks in size. Since in the closed cranium the brain cannot shrink, the cerebral capillaries would be congested with the venous at the expense of arterial blood, if the cerebral arteries ever became constricted; and this fact may in itself explain the lack of cerebral vaso-motor action. There is no need to evoke a cerebral vaso-motor mechanism on behalf of the doctrine of cerebral localisation; for, psychologically, it is impossible to maintain that any one portion of the brain can act alone. So long as the mind is in activity, thought skips from memory to memory, while sensations stream from every sense organ to every sense centre. The cerebral circulation is regulated by the action of the cardio-inhibitory and vaso-motor centres; in what manner, will be shown later.

When the arterial pressure rises, the expansion of cerebral volume can take place to a certain small amount only. For, as soon as all the cerebrospinal fluid has been driven out of the meningeal spaces, the brain comes everywhere in contact with the rigid wall of the skull. Any further expansion of the arteries and capillaries can only take place by an equivalent compression of the veins, for the semi-fluid brain matter is incompressible. The reservoirs of blood in the veins will therefore be constricted until the cerebral venous pressure again becomes equal to the intracranial pressure; that is, to the pressure of the brain against the veins. Then the whole circulatory system of the brain will have assimilated itself to a scheme of rigid tubes; the velocity of the blood-flow will be increased, while the relative distribution of the blood in the arteries, capillaries, and veins will be changed. The doctrine has been enunciated that intracranial pressure varies as the tension of the cerebral arteries, and that, consequently, a high pressure in these arteries must lead to decrease in the amount of blood flowing through the brain. This doctrine is entirely erroneous. The intracranial pressure is found in all physiological conditions to be the same as the cerebral venous pressure. The intracranial pressure is that which remains after the force of the heart has been expended in driving the blood through the capillaries. It is therefore the same as the pressure in the venous sinuses. If the intracranial pressure becomes pathologically greater than the pressure in the venous sinuses then the cerebral circulation is disordered. By no physiological means can intracranial pressure be maintained at a higher level than cerebral venous pressure; because (*a*) directly the capillaries and veins are obliterated the pressure rises in them to the static pressure, that is, to the mean arterial pressure; (*b*) arterial pressure, transmitted directly through the arteries to the capillaries and veins, must always be greater than arterial pressure minus the elastic tension of the arterial wall transmitted through the brain substance. So long as the high arterial tension lasts, the expansion of the cerebral vessels reaches its utmost limit, they approach the condition of rigid tubes, and the velocity of the circulation is greatly increased. Thus during the great rise of arterial pressure produced by the injection of absinthe the outflow of blood from the torcular has been seen to increase from two to six times.

Intracranial pressure is purely circulatory in origin, and therefore is variable in quantity. By the term intracranial tension is signified the pressure which the surface of the brain exerts against the walls of the cranium. Cerebral pressure is an expression which has an old pathological connotation, and in dealing with physiological facts it is necessary to avoid its use. Intracranial pressure is the same as cerebral venous pressure, and varies in the same direction absolutely as vena cava pressure; proportionately as general arterial pressure.

If, in the living animal, the cerebral venous and intracranial pressures be recorded simultaneously with the vena cava and aorta pressures, and if the abdomen be compressed at the same time, the pressures in the brain and the vena cava rise by equal increments, when, that is to say, the arterial pressure remains constant; and by greater increments in the brain if the arterial pressure rise also. While the intracranial and cerebral venous pressures vary absolutely as the pressure in the venæ cavæ, they do not vary absolutely as the general arterial pressure, but only in the same direction, because between arteries and capillaries there lies the unknown resistance in the arterioles due to the viscosity of the blood. In the brain, as in other parts of the body, variations of arterial pressure, read in millimetres of mercury, only produce variations in cerebral venous pressure which can be read in millimetres of water.

The cerebro-spinal fluid pressure does not by any means always correspond with the intracranial pressure. For the brain can expand to a far greater degree than the spinal cord, and the brain on expansion, after expressing the small quantity of cerebro-spinal fluid from the cranium, comes into contact with the cranial wall. On the other hand, the cerebro-spinal fluid not only distends the vertebral ligaments, but leaks away from the subarachnoidal space.

While in normal conditions, with the animal horizontal, the intracranial and cerebral venous pressures generally equal about 100 mm. H_2O, there is no compensatory mechanism by which the brain matter can be protected from great changes of circulatory pressure.

During forced expiratory effort, when the outlets of the venous blood into the thorax are blocked by the high intrathoracic pressure, intracranial or cerebral capillary tension must almost rise to the arterial tension. In the spasms of strychnine poisoning the intracranial pressure has been observed to rise to 50 mm. Hg.

On the other hand, in a certain patient, I have observed the intracranial tension to be slightly below zero while he was standing upright. This patient had been trephined for symptoms of insanity. It is thus clear that the functions of the brain can continue when that organ is submitted to very large variations of pressure. In these respects there is nothing which pertains very peculiarly to the cerebral circulation. The circulation within the marrow of the bones must be under exactly the same conditions as that of the brain. The limbs, again, are encased with skin which allows only a certain definite amount of expansion; when this limit is reached any further congestion of blood, such as follows forced expiratory effort, is prevented, and the tissues of the limb must be submitted to almost static arterial tension. Since the soft parts of the body yield and expand to arterial pressure to a large degree, determination of blood from one part to another can be there brought about by means of the vaso-motor mechanism. In the brain, not the blood quantum, but rather the velocity of blood flow is increased, whenever the arterial pressure is raised by general vaso-constriction.

Since the cerebral circulation is uncontrolled by any local vaso-motor mechanism, and passively follows every change in the general circulation, it is necessary to determine how the latter maintains the efficiency of the former. This leads us to the discussion of a matter of great importance in cerebral pathology, namely, the effect of the position of the body on the circulation.

In a man, 6 ft. high, the hydrostatic pressure of a column of blood reaching from the vertex to the sole of the feet equals 140 mm. Hg; and from the vertex to the middle of the abdomen about 50 mm. Hg. If in a dog the heart be inhibited, after the skull has been opened, and the animal be placed in the vertical feet-down posture, the brain and heart will empty of blood, while the splanchnic veins and capillaries become distended. In the feet-up posture these conditions will be reversed.

If, on the other hand, the heart be at work, and the animal be placed in the feet-down position, the circulation through the brain ceases whenever the power of the cardiac and respiratory pumps is not sufficient to raise the blood from the splanchnic area, to lift it to the brain, and to overcome the resistance in the cerebral arterioles. In the vertical feet-down position the pressure in the splanchnic and leg areas is increased by the height of the column of blood, which extends from the brain to these vascular areas. This tends to enlarge their capacity; to these parts the flow of blood is aided, while that to the brain is opposed.

Now in man the column of blood on the venous side is broken into short segments by valves in the veins of the arms, the jugular veins, and the veins of the legs. The longest column of venous blood, unbroken by any valve, is that which extends from the superior longitudinal sinus, by way of the anastomosis with the intravertebral sinuses, to the azygos veins, and thence to the end of the vena cava inferior. Symington and others have thrown doubt on the competency of the jugular valves. The function of the valves is to prevent the over-distension of the lower veins by gravity, and to allow the venous blood to be returned to the heart by the movements of the skeletal muscles. In pathological states leading to incompetency of the valves over-distension is seen to arise and to produce varicose veins.

The capacious abdominal veins are supported under the weight of the column of blood by means of the abdominal wall. The efficiency of this support depends on the tone of the muscular wall and the integrity of the respiratory centre. The veins of the limbs are supported by the muscles on the one side, and the external sheath of the limb—the skin—on the other side. By such means the distension of the veins and capillaries in the erect posture is largely prevented. The determination of the blood into the lower parts of the body under the influence of gravity is largely prevented by the contraction of the skeletal muscles, by the peristalsis of the viscera, and by the action of the respiratory pump. The respiratory movements aid the venous flow by the alternate compressive action of the abdominal wall and suction action of the thorax. On the arterial side of the circulation the arterioles are held in a state of tone by the vaso-motor centre, and limit the output into the veins.

When an animal is suddenly turned from the horizontal to the vertical feet-down position, the peripheral resistance in the great splanchnic area is increased, and the capacity diminished; thus the circulation through the head is maintained. The blood in all cases takes the pathway of least resistance. The great splanchnic area forms the resistance box of the circulation; if its channels be constricted the blood takes the pathway through the locomotor organs; if they be dilated the blood accumulates in the capacious veins of the abdomen. When the splanchnic area is fully dilated the whole of the blood collects within the abdomen, and, if the animal be in the vertical feet-down posture, the influence of gravity may rapidly bring about this result. Splanchnic vaso-motor paralysis can, however, be compensated by the action of the respiratory pump. The capacity of the abdominal vessels can be reduced by the compressive action of the respiratory muscles, and the output from the splanchnic arterioles held in check; while the blood pressed up the vena cava can be aspirated into the right heart by the inspiratory action of the thorax.

If the spinal cord be divided at the level of the first dorsal vertebra, the influence of the bulbar centres on the parts below the section is removed, abdominal and intercostal breathing cease, the respiration becomes purely diaphragmatic, the tone of the great splanchnic area of the arterioles is lost, and the capacity of the abdominal vessels is greatly increased. The total effect on an animal, when lying in the horizontal posture, is a considerable fall of the arterial pressure, coupled with a marked diminution of the respiratory oscillations. If the animal be now placed in the vertical feet-down position the arterial pressure falls more rapidly, and the circulation may come to an end. This is so because the blood passes through the dilated arterioles and collects in the abdominal veins; no mechanism, except the diaphragm, is left for filling the right ventricle, and thus the heart, empty of blood, continues to beat to no purpose. If now the abdominal wall be firmly compressed with the hand, the capacity of the splanchnic area is reduced, the right ventricle filled with blood, and the circulation renewed. To sink the animal in a bath has the same effect. On taking off the hand the heart empties again, the arterial pressure falls, and the circulation ceases. When the animal is once more placed horizontally the influence of gravity is abolished, and the circulation immediately becomes re-established. Experiment shows that the respiratory pump can compensate gravitation when the tone of the splanchnic area is destroyed by section of the splanchnic nerves. The respiratory pump, on the other hand, can be paralysed by the injection of curari. Under these conditions the heart is able to maintain the circulation so long as the capacity of the abdominal vessels is kept under control by the vaso-motor nerves.

Whenever, by any means, the thoracic pressure is rendered sufficiently positive to prevent the diastolic filling of the heart the blood stagnates in the abdomen and the circulation ceases. Owing to the influence of gravity this state of affairs is brought about more easily in the vertical feet-down position than in the horizontal posture. Thus when an animal is in the feet-down position the circulation can be easily stopped by injection of fluid into the pericardium by compressing the thorax, or by forcibly expanding the lungs with air. In the crushes of panic-stricken crowds death takes place from syncope induced by compression of the thorax and cessation of the cerebral circulation. The importance of the respiratory pump as an aid to the circulation is shown by the symptom of orthopnœa seen in cardiac and pulmonary disease. The patient does not struggle for air, but struggles by means of his respiratory efforts to maintain a sufficient circulation in the face of cardiac defect, or of increased pulmonary resistance.

On the assumption of the feet-up posture there exists no great vascular field like the splanchnic area to expand under the weight of the column of blood. The face and neck alone become congested, for the brain is confined within the rigid wall of the cranium. In the feet-up posture the arterial pressure in the carotid artery is kept down by a compensatory mechanism. The heart is slowed; the action of the respiratory pump is diminished; the output of the heart is lessened; possibly dilatation of the splanchnic arterioles also takes place.

It is apparent that there exists some special mechanism by which the pressure in the carotid artery, and thus of the cerebral circulation, is maintained practically constant during changes of posture. The nervous mechanism involved is probably of the simplest kind, for, if the arterial pressure suddenly rise or fall, the bulbar centres are thereby either directly or reflexly excited to decreased or increased activity. A sudden fall of arterial pressure always provokes acceleration of the heart, amplified respiration, and increased vaso-constriction. A sudden rise of pressure, on the other hand, provokes a slow heart, shallow respiration, and vaso-dilatation.

When the compensatory mechanism is abolished by destruction, exhaustion, or inhibition of the bulbar centres, the circulation fails in the vertical feet-down position, the blood passes into the abdomen, and the cerebral circulation ceases. There can be no doubt that the control of this compensatory mechanism is one of the most important and absolutely necessary functions of the group of bulbar centres, a function which must be evolved to its highest point in man.

That the influence of gravity has something to do with the causation of emotional syncope is clear from the success which follows lowering of the patient's head between the knees. Syncope is commonly regarded as being due to a cessation of the action of the heart. It is, however, well known that in cases of syncope the heart does not cease to beat, but continues to pulsate rapidly and very feebly.

The symptoms of syncope are exactly similar to those conditions observed in an animal placed feet downwards with the compensatory mechanism for gravity abolished. By sudden fright in the standing posture the action of the respiratory and vaso-motor centres is inhibited, and syncope is induced by the rush of blood to the abdomen and cessation of the cerebral circulation. The patient is restored on being placed in the horizontal or feet-up position. A deep sigh is the first sign of improvement; the right heart is thus filled with blood by the thoracic suction which is accompanied by retraction of the abdomen.

Patients who have lain long in bed, especially when exhausted by disease, lose the power of adapting themselves to changes of posture. Hence the dizziness and danger of syncope which occur when the patient first rises from bed. The condition of shock may be largely, if not entirely, explained by the loss of the compensatory mechanism which maintains the circulation in the face of the influence of gravity. A condition of shock always advances slowly during the course of a prolonged experiment on an animal. The arterial pressure steadily falls; the respiratory oscillations of pressure become less; the heart-beats weaken as the coronary circulation becomes less efficient; the temperature of the animal decreases; the fall of pressure in the feet-down position becomes steadily greater. Such a condition is rapidly intensified if injury be accompanied by severe hæmorrhage (*vide* art. "Shock and Collapse," vol. iii. p. 320).

Among drugs in common use chloroform stands prepotent as a drug which has the power to abolish the compensatory mechanism. Chloroform causes cardiac and vascular dilatation by acting directly on the musculature of the heart and vessels, weakens the respiration, and abolishes the tone of the abdominal and skeletal muscles. Cases are occasionally recorded of men, some of whom lose their memory in the upright posture, and regain it when in recumbency; others can only do mental work when in the horizontal posture; others are suddenly at a loss for memory when attempting to speak in public. These symptoms, and similarly the effects of fear which are so commonly manifested by men before battles, examinations, or public appearances—the vomiting, diarrhœa, and involuntary micturition—may all be associated with temporary inhibition of the compensatory mechanism, and determination of blood to the abdomen.

A useful clinical guide to the condition of the compensatory mechanism in man is afforded by the frequency of the pulse on change of posture. If the heart be markedly accelerated on suddenly passing from the horizontal to the vertical position the mechanism is inefficient. In some cases the change of pulse-rate may be as much as 20 to 40 beats per minute; normally it does not exceed 5 to 10 beats per minute.

The arterial pressure in man can be accurately taken by means of the Hill-Barnard sphygmometer.[1] In its simplest form this sphygmometer consists of a vertical glass tube six inches in length. The tube ends above in a small bulb; below it expands into a small shallow cup. The cup is closed by a flaccid rubber membrane, and is filled with coloured fluid. On pressing the cup down over the radial or other artery the fluid rises in the tube and compresses the air in the bulb. The air acts as an elastic spring. At a certain pressure-height the fluid meniscus exhibits maximal pulsation. The tube is graduated in mm. Hg; and, to avoid errors due to barometric and temperature changes, the fluid meniscus can by a special mechanism be set at the zero of the scale before each observation. So soon as the mean pressure within and without the artery is the same, the wall of the artery oscillates with the greatest freedom at each systole of the heart. At this point the pressure is read, and the reading is the mean arterial pressure. It has been proved that the maximal excursion is an accurate index. In the normal healthy individual the mean pressure in the brachial artery is the same in the horizontal as in the erect postures, or it may be slightly higher in the latter position. Thus the compensation for the effect of gravity is perfect.

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[1] Made by Hicks, 8 Hatton Garden, E.C.

In states of neurasthenia and exhaustion the pressure may be less in the erect posture, and is maintained by a greatly accelerated heart-beat. It is obvious that in such states the maintenance of the horizontal position, and the wearing of an abdominal belt, will aid the circulatory mechanism. The effect of change of posture is the clinical key to the condition of the vaso-motor mechanism. In the erect posture the pressure in the tibial arteries is higher than that in the brachial, by the height of the column of blood which separates these arteries. In the horizontal posture the pressures are the same.

From the above discussion it is now clear that the splanchnic vaso-motor mechanism is of overwhelming importance in maintaining the cerebral circulation. We have, in the vaso-motor centre, a protective mechanism by which blood can be drawn at need from the abdomen and supplied to the brain. The respiratory centre renders important aid. At the moment that excitation from the outside world demands cerebral response, the splanchnic area is diminished, and more blood is driven through the brain. An anæmia of the brain excites the bulbar centres; these provoke acceleration of the heart, splanchnic constriction, and increased respiration; the cerebral circulation is thereby restored. The brain has no direct vaso-motor mechanism; but its blood-supply can be controlled indirectly by the bulbar centres acting on the splanchnic area.

These centres are part of the central nervous system, they feel the same needs, and are stimulated by the same centripetal impulses as affect the rest of that system; thus a supply of blood to the brain is maintained which corresponds to its functional activity.

From this section we may conclude that :—

1. No conclusive physiological evidence has been found which points to the existence of cerebral vaso-motor nerves.

2. In every experimental condition the cerebral circulation seems passively to follow the changes in the general arterial and venous pressures.

3. The intracranial pressure, or the pressure of the brain against the cranial wall, is in all physiological conditions the same as the cerebral venous pressure.

4. The intracranial and cerebral venous pressures vary directly and

absolutely with vena cava pressure, but only proportionately with aortic pressure.

5. The intracranial pressure is purely circulatory in origin, and may vary largely with the changes in circulatory pressure. Normally, in the horizontal posture, it equals about 10 mm. Hg.

6. The volume of blood in the brain is, in all physiological conditions, but slightly variable. The venous side of the cerebral circulation may, however, be congested at the expense of the arterial side, or the arterial side at the expense of the venous.

7. In all physiological conditions a rise of arterial pressure accelerates the flow of blood through the brain, and a fall slackens it. A rise of vena cava pressure impedes the cerebral circulation.

8. The cerebral circulation is controlled by the bulbar centres which act on the general circulatory system. When the brain requires more blood the heart is accelerated, the splanchnic arterioles constricted, and the pressure in the carotid artery raised.

9. The force of gravity must be regarded as a cardinal factor in dealing with the circulation of the blood.

10. In the upright position the effect of gravity is compensated by means of the cardiac acceleration and the vaso-motor mechanism. The pressure in the carotid artery is thus maintained. From the lower part of the body the blood is returned to the heart by means of muscular movements acting on the valved veins, and by the action of the respiratory pump.

11. When the power of compensation is damaged by loss of muscular tone and paralysis of the vaso-constrictor mechanism, induced by shock or exhausting disease, the influence of gravity becomes of vital importance. In the upright posture the blood then drains into the great abdominal veins, the heart empties, and the cerebral circulation ceases.

12. The horizontal and feet-up postures at once abolish syncope induced by the feet-down posture, by causing the force of gravity to act in the same sense as the heart, whereby the cerebral circulation is renewed. Firmly bandaging the abdomen has the same effect.

13. If the heart be weakened by some poison, such as asphyxial blood or chloroform, the sudden dilatation of the right ventricle produced by the assumption of the feet-up position, or by pressure on the abdomen, may throw the heart into paralytic dilatation.

14. Paralytic dilatation of the heart is relieved by the upright posture, coupled with rhythmic compression of the thorax. The right ventricle is thereby emptied into the abdominal veins.

15. During changes of posture the heart frequency is normally but little altered (5-10 beats). When the compensatory mechanism is defective, the frequency may be greater, by 30 to 40 beats, in the upright than in the horizontal position. This is a clinical index of defective compensation.

16. The cerebral circulation is lessened by anything that obstructs the pulmonary circulation. A rise of intra-thoracic pressure impedes the

flow of blood through the lungs, lowers the arterial pressure, raises the vena cava pressure, and congests the brain with venous blood. Thus syncope can be easily produced by forcible compression of the thorax.

17. Similarly, the cerebral circulation will be impeded by any valvular defect of the heart, which raises the general venous pressure; for the rate of flow through the cerebral capillaries depends on the difference of pressure between the cerebral arterioles and cerebral veins.

Cerebral hyperæmia.—On the grounds of experimental research, it has been made evident that no noteworthy increase in the quantity of blood within the cerebral capillaries can take place suddenly. But an increased flow of blood through the brain is produced whenever, by forcible action of the heart or contraction of vascular areas, the general arterial pressure is raised. The relative amount of arterial and venous blood within the brain may then alter—that is to say, the arterial side of the cerebral system may be more, and the venous side less distended than normal. In passive venous congestion of the brain just the opposite condition will hold.

The term cerebral hyperæmia can be used to express that condition in which the flow of arterial blood through the brain is increased per unit of time. From such hyperæmia nothing but good to the organism should arise. By the rise of arterial pressure which takes place in outdoor exercise the brain is swept with a stream of arterialised blood, and exhilaration and mental vigour results. In times of mental labour the same thing happens. The sphygmometer shows that the arterial pressure is raised by mental labour, as during the giving of a public lecture, and throughout the course of an animated argument. This arterial hyperæmia is primarily the sequence and not the cause of functional activity in the brain. Sensory excitement awakes the dormant activities of the cerebral centres, and the circulation is braced up for the struggle. It is necessary to remember that, we have no means of analysing the effect of the nerve impulses which stream in upon the centres from the effect of the blood-supply on these centres. During sleep impulses cease to excite, and the brain is refreshed by rest; at the same time, the sphygmometer shows that the arterial pressure is lowered, the cerebral veins are congested by the horizontal posture, respiration is far less deep, and the plethysmograph records that the blood is derived in increased volume to the limbs. Thus the brain tissue recovers from fatigue in spite of a diminished cerebral circulation. The expansion of the vessels of the limbs is due probably to two causes: (i.) warmth; (ii.) diminished respiration and absence of muscular movement. Owing to the former the vessels dilate; owing to the latter the blood congests in the veins. As we awaken the blood forsakes the periphery; by the increased action of the heart, the greater tone of the arterial system, and the important aid which respiration and muscular movements render to the venous circulation, it is driven in a greater volume through the brain. We cannot say that either the wakening or the sleeping state is produced by these circulatory changes. I have observed a patient to fall asleep while the arterial pressure remained constant; the fall indicated by the sphygmometer may be as great during a period of rest as during sleep. In sleep, as in chloroform anæsthesia, the brain is congested with venous blood at the expense of arterial blood. It is, however, more probable that this is the result rather than the cause of the functional inactivity of the nervous system. Soon after a short period of hard muscular exercise the sphygmometer shows a fall of arterial pressure. This fall follows the rise of pressure which is found during the period of exercise, and is due probably to the vascular dilatation of the skin and muscles. In spite of this fall of pressure the brain is then often most fitted for mental labour.

Writing of men of genius, Lombroso says that "some, in order to give themselves up to meditation, even put themselves artificially into a state of cerebral semi-congestion. Thus Schiller plunged his feet into ice. Pitt and Fox prepared their speeches after excessive indulgence in porter. Paisiello composed beneath a mountain of coverlets. Descartes buried his head in a sofa. Bossuet retired into a cold room, with his head enveloped in hot cloths. Cujas worked lying prone on the carpet. It was said of Leibnitz that he 'meditated horizontally,' such being the attitude necessary to enable him to give himself up to the labour of thought. Milton composed with his head leaning over his easy-chair. Thomas and Rossini composed in their beds. Rousseau meditated with his head in the full glare of the sun. Shelley lay on the hearthrug with his head close to the fire." It is possible that some of these men, by means of ice to the feet, or a cold room, increased the flow of arterial blood through the brain, just as many men can think best when exhilarated by rapid exercise, or when walking up and down a room. On the other hand, lying in bed under a mountain of coverlets, or excessive indulgence in porter, lowers the arterial tension. In some cases where the circulation is feeble and the compensation for gravity defective, active thought may only be possible in the horizontal posture. These oddities of genius cannot be explained thus lightly on purely mechanical grounds; nor must we forget that postural tricks are common enough in ordinary men and under ordinary circumstances.

Arterial hyperæmia is probably always the sequence and not the cause of a primary functional activity in the central nervous system. The arterial pressure is raised by all conditions which produce mental excitement.

In animals arterial hyperæmia of the brain, produced experimentally, gives rise to no symptoms. "Of all regions of cerebral pathology," writes Sir R. Gowers, "that of congestion of the brain is most obscure. We have very little precise knowledge regarding it; and as is often the case, theory has flourished in proportion to the deficiency of fact. It was long thought that the state of the vessels of the brain after death corresponds with their condition during life, and post-mortem distension was accepted as proof that any preceding cerebral symptoms were due to congestion. The fact was unobserved or ignored that a similar condition of the brain is equally common when there are no cerebral symptoms during life, and depends chiefly on the mode of death." It is highly probable that many symptoms have been erroneously ascribed to cerebral hyperæmia which should be placed at the door of cerebral venous congestion, or of anæmia of the brain. Venous congestion, as will be shown later, is of the utmost pathological importance; for any increase of general venous pressure, by impeding the flow of blood through that organ, will tend to produce cerebral anæmia.

I am inclined to deny the existence of simple hyperæmia of the brain as a pathological state. Heightened arterial blood-pressure is probably, in all cases, merely a symptom of that pathological condition by which the other symptoms of cerebral disturbance are produced. It is to be particularly remembered that the vascular condition of the face and head and neck does not necessarily correspond with that of the brain For example, the flushing, the throbbing, and the headache produced by amyl nitrite are associated with dilatation of the heart and vessels, a fall of arterial pressure, and a greatly diminished cerebral circulation.

In states of over-fatigue, or of mental excitement, the arterial blood is, according to sphygmometer readings, run at high pressure, while the frequency of the heart is notably accelerated. After any severe effort the circulatory mechanism takes time to return to its normal quiet level. The cerebral excitement starts the energetic circulation, but the latter adds fuel to the former, and continues to maintain the brain in activity when the time for effort has ceased and repose should come. In this condition, when sleeplessness and mental irritability torment the exhausted worker, it is well known that diversion of blood to the body by hot baths, hot drinks, and warm bottles, by raising the head high, and by purgation, are simple and effective forms of treatment. Even in these cases it may be the soothing nature of the nerve stimuli, rather than any alteration in the circulation, that induces sleep. Cold, when applied to the head experimentally, produces no effect on the cerebral circulation. The employment of the wet towel by the student who burns the midnight oil must therefore find its explanation in some stimulation to the brain, which is of sensory and not circulatory origin. It is possible that the cerebral tissue is actually cooled below the body temperature by the application of an ice-cap. This cooling may produce the required therapeutic effect.

From this section it may be concluded:—(i.) Arterial hyperæmia of the brain does not exist as a pathological state. (ii.) Sphygmometer readings of arterial pressure in man show that the tension of the cerebral arteries is constantly varied by change of external temperature, exercise, sleep, baths, food, etc. Such variations are purely physiological. (iii.) There is every indication that the quality of the blood which flows through the brain is of far greater importance than the quantity. (iv.) Mental exhaustion probably does not arise during the stage of hyperæmia produced by continued over-effort, but supervenes at that period when the circulatory mechanism fails, and venous congestion is the fate of the exhausted cerebral centres.

Cerebral anæmia.—The effects which follow ligature of the common carotid arteries have been known from classical times. The very name of the artery betokens sleep. "Peripatetic mountebanks used," as we are told, "to include a goat among their stage properties, and were said to tie up and relax these arteries in the animal, so that at their pleasure the goat fell down motionless and stupid, or at their bidding leapt up again with great vigour."[4] The first important experiment on this subject was that of Astley Cooper. He occluded both the carotid and vertebral arteries in a rabbit; spasms immediately resulted, and the respiration ceased. In a dog Cooper ligatured the same four arteries. After a preliminary stage of paralysis the animal recovered, and lived to be an excellent house dog. Kussmaul and Tenner tied the left subclavian and innominate arteries. The immediate symptoms were loss of consciousness and voluntary movement. These were followed, in ten to forty-five seconds, by clonic spasms beginning in the muscles of the neck; then occurred dilatation of the pupils, respiratory gasps at longer and longer intervals, and finally cessation of respiration. After complete occlusion of these arteries, for not longer than two to three minutes, the brain, on the ligatures being once more loosened, showed the power of complete recovery. The sudden re-entry of blood stopped the spasms, and in no case did it cause them. On loosening the ligatures the rabbit often gave a sudden jump forward, then remained for a few moments as if paralysed, and finally recovered completely. Death without spasm occurred in debilitated or over-anæsthetised animals. These spasms are not occasioned by the change of intracranial pressure, for they are produced in no less a degree when the pressure is maintained by an injection of normal saline solution into the carotid artery. The spasms and other symptoms likewise appear when all the cerebral venous outlets are blocked. If one venous outlet be left unobstructed an efficient circulation is maintained, and the animals do not die.

Couty, Markwald, and others have produced anæmia of the brain experimentally by the method of embolism. Either fine powder or melted wax has been used to plug the cerebral arteries. The arrest of the circulation of the whole brain by embolism almost immediately causes cessation of respiration, an enormous rise of arterial pressure, and slowing of the heart-rate. The heart is sometimes completely inhibited. In a few minutes death follows, preceded by acceleration of the heart and fall of pressure. Embolic anæmia, of the cerebral hemispheres alone, causes slowing of the pulse, with no rise of arterial pressure. The animal is sleepy and unable to walk, while the limbs remain in abnormal positions. There is no response to stimulation of light or sound. If the mid-brain be cut off from its blood-supply opisthotonos occurs. If the basilar artery be embolised by injections into the vertebral arteries, the greatest pressor effects occur, and after some spasms from failure of respiration death quickly results

The effects which follow ligation of the four cerebral arteries vary greatly in different animals; to a less extent in different individuals. In dogs a free anastomosis is set up by way of a branch from either superior intercostal artery. This passes in with the brachial plexus and joins the anterior spinal artery. These arteries, after the ligature of the vertebrals and carotids has been carried out, become almost as large as the vertebrals, and maintain an efficient circulation. Thus, in dogs, death never ensues. On the other hand, almost all rabbits and 40 per cent of cats die, either at once or in a few hours after the quadruple ligature has been simultaneously applied. Monkeys likewise die in the course of a few hours. Most monkeys survive the simultaneous ligation of both carotids. They usually succumb to ligation of the two carotids and one vertebral. If the ligatures be applied in monkeys, not simultaneously but successively, at intervals of a few days, no untoward symptoms arise. When a record of cerebral venous pressure is taken and the four cerebral arteries are ligatured in the dog, the venous pressure is seen at first to fall distinctly, and then gradually to recover itself. The pressure in the circle of Willis (peripheral end of carotid) falls likewise. It rises subsequently as the anastomotic pathways become open, and even in some cases to a higher level than normal. This is owing to the pressor effect produced by bulbar anæmia.

The symptoms of acute cerebral anæmia are the same whether produced by ligature of the arteries or by embolism. If the cerebrum be rendered bloodless, loss of consciousness and general motor paralysis result. Epileptiform spasms may occur in strong subjects. The pupils dilate and nystagmus often occurs. Rigidity and reflex hyper-excitability are marked symptoms.

In monkeys if two carotids and one vertebral be ligatured, the pupil is dilated on the side opposite to the obstructed vertebral. When the hind brain is suddenly rendered anæmic, there ensue general spasm of a tonic character associated with rigidity, vaso-motor spasm producing a high blood-pressure, respiratory spasm, and a slow heart caused by spasm of the vagus centre. The state of spasm is followed by paralysis, as is shown by the failure of respiration, fall of arterial pressure, and acceleration of the heart. In exhausted or deeply anæsthetised subjects spasm does not appear, and the stage of paralysis is rapidly reached. Generally the primary cause of death is failure of respiration. If this be compensated by artificial respiration the heart continues to beat, and then the vaso-motor paralysis which ensues may be a secondary cause of death. This can be compensated by compression of the abdomen and by placing the animal in the vertical feet-up position. In rare cases vaso-motor paralysis may be the primary cause of death.

Cheyne-Stokes respiration and Traube-Hering blood-pressure curves are commonly obtained in conditions of anæmia of the spinal bulb. The injection of morphine, a solution of magnesium sulphate, and the administration of chloroform frequently produce these curious phenomena in dogs, especially after ligation of the cerebral arteries. In hybernating animals they are constantly present; group respiration may frequently be seen in sleeping children. The phenomena may occur when the arterial pressure is high. Such is the case in dogs poisoned with morphine. In some cases Traube curves may be peripheral and not central in origin. This periodicity of action seems to be a sign of lowered nutrition in the bulbar centres.

Obliteration of the cerebral arteries in man produces the same symptoms as in animals. If both common carotids be suddenly compressed, the pupils widen, respiration deepens, dizziness and loss of consciousness follow. Epileptiform spasms frequently occur. I have produced clonic spasms in myself by compression of one carotid artery, thus confirming the experience of Schiff. The first effect of the compression was a tingling sensation in the eye on the same side; then followed a march of a sensation of pins and needles down the opposite side of the body: this began in the fingers, spread up the arm, then down the leg. Finally, clonic spasms of the flexors of the forearm occurred, accompanied by a feeling of vertigo and alarm. Consciousness of the clonus was aroused by the sensation of the hand striking the arm of the chair. In dogs, on the other hand, with the four cerebral arteries tied, I find the excitability of the cortex markedly increased.

The effects of compression of one carotid vary in different men; no doubt in relation to the freedom of the anastomosis in the circle of Willis, and to the excitability of the cerebral cortex. The spasms, when produced, are in every respect the same as Jacksonian epilepsy, or the epileptic fits produced by tetanisation of the sensori-motor area. On occlusion of one carotid artery in the monkey compensation by the circle of Willis is not immediately complete. Horsley and Spencer found that, on ligaturing one carotid, the pia mater pales on the side of the ligature, and the cortex becomes far less excitable. The first effect of such anæmia is to raise the excitability of the cortex (Orchansky). In an hour the circulation is fully restored. From the results of physiological experiment we can be assured that ligature of both carotids, if done slowly, and at due intervals of time, can be carried out without fatal results. If the artery were so gradually occluded by a screw clamp as to allow time for expansion of anastomotic pathways, no ill effects, such as hemiplegia, should ever result. There is a pathological case on record of gradual obliteration of all four cerebral arteries; efficient anastomosis was established in this case, and no cerebral symptoms were evoked during life.

Sudden and simultaneous ligature of both carotids must be regarded as a highly dangerous operation. Sudden occlusion even of one carotid has in some cases been followed by hemiplegia.

In certain cases, by the simultaneous ligation of the four arteries in the dog, or of three arteries in the monkey, a condition of idiocy has been established. To produce this is not easy, as the cerebral circulation must be reduced to such a point that the animal hangs between life and death. Most dogs recover from the ligation of the two carotids and two vertebrals, after but slight and temporary signs of paralysis. In some cases one superior intercostal artery has been tied also, and yet the animals have completely recovered. Most monkeys, on the other hand, die in the course of twenty hours from the ligation of two carotids and one vertebral. In one case the animal lived in a condition of complete idiocy, paralysis and rigidity. When in another monkey one carotid and one vertebral were tied, and the other carotid ligatured after an interval of three days, no marked symptoms ensued. Thus a very few days is sufficient to establish the expansion of anastomotic pathways.

A dog rendered idiotic by the method of ligation exactly resembles Goltz's brainless dogs. He sleeps and wanders round the room alternately; there is a continual tendency to circus movements; the tactile sense is greatly paralysed; movement is defective; his legs slip outwards and give way under him. He stumbles blindly into every object, but can smell and hear. An enraged cat, tobacco smoke, lighted matches, pass equally unnoticed before his face. He persistently tries to get through impossible objects, and finally falls asleep in front of them, his body and limbs being placed in unnatural positions. He eats reflexly when food is placed in his mouth. The optic discs present congested veins and small anæmic arteries.[1] In four or five days' time all these symptoms disappear, and the animal recovers completely.

On histological examination of the brains of these dogs, Dr. Mott finds that within twenty-four hours of the operation, and during the stage of idiocy, the nuclei of the cortical cells become swollen, and the cytoplasm of the cell is stained diffusely by Nissl's methylene blue method. The stichochrome granules disappear from the cells, to reappear once more when the animal recovers. In the monkey the pathological changes have gone further, and the symptoms have been more profound. One monkey became completely rigid, in the extended position, anæsthetic and paralysed. It remained in the same condition for five days and was then killed. The brain was softened. Microscopically, many of the cortical cells were found greatly swollen and devoid of stichochrome granules; the perivascular spaces were distended with exudation, and contained numerous phagocytes. It is noteworthy that by Golgi's method the cells and their dendrites appeared unaltered. It is extraordinary that this monkey, although dead to all other forms of stimulation, when a cat entered the room twice made a hoarse guttural sound of alarm and attempts at flight movements.

Cerebral anæmia of slow onset.—When general anæmia of the brain comes on gradually, symptoms of exalted excitability do not appear. In man, headache, hyperæsthesia tinnitus, and vertigo are common symptoms, associated with mental dulness and drowsiness. In some cases great mental irritability and insomnia may appear. The patient loses the power of decisive action, may suffer from melancholia and be filled with unreasoning fears. Dimness of sight or double vision and deafness may be complained of. Muscular power is weakened, memory is deficient, power of application and energy of action are lost. Continued anæmia of the brain may result from heart failure, from loss of vaso-motor tone, from exhausting diarrhœa, or from defects in the quality of the blood.

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[1] In these conditions the author finds the cortex to be *hyper-excitable*. The sensory and not the motor pathways are disturbed.

Whatsoever tends to raise the general venous pressure causes at the same time cerebral venous congestion; and this, by obstructing the flow of arterial blood, leads to anæmia of the brain. Likewise whatsoever tends to lower arterial blood-pressure lessens the flow in the cerebral arteries.

In infants collapsed from choleraic diarrhœa the depression of the fontanelle shows that the blood-pressure is insufficient to overcome the resistance in the cerebral arteries. In these cases pallor of the face is well marked. Somnolence may pass into coma, then the pupils dilate, and become insensible to light; and death results from the gradual paralysis of the bulbar centres.

Chronic anæmia of the brain may result in permanent damage. In children the development of the brain may be arrested, and imbecility be the consequence. Long-continued general loss of vascular tone or degeneration of cerebral arteries, and consequent failure of efficient cerebral circulation, may be a fruitful source of cerebral degeneration and of lunacy. Valvular disease of the heart may lead to the same end. Persistent mechanical congestion of the veins, arising from pulmonary disease and from cough, must reduce the cerebral circulation. The recumbent posture is the obvious treatment in simple cases of cerebral anæmia. Sudden changes of position should be avoided. In all conditions of exhaustion of the circulatory mechanism absolute rest in bed is most beneficial. The period of complete rest should be followed by gradual return to periods of gentle exercise. Strychnine is indicated, and, in conditions of restlessness and insomnia, morphine. It should be remembered that morphine is one of the best vaso-constrictors and cardiac tonics we possess.

General tonic treatment should be employed, as it must always be borne in mind that the deficient circulation may be more often a symptom than the primary cause of nervous exhaustion.

The fact that the brain can continue its functions with a greatly reduced blood-supply is shown, not only by the experiments on the effect of ligation of the cerebral arteries, but by the lack of any marked cerebral symptoms in advanced cases of pernicious anæmia. It has been demonstrated by determination of the gases in cerebral arterial and venous blood that the brain is not a seat of active combustion. The combustion of the muscles, whether at rest or in the state of epileptic spasm, is vastly greater than that of the brain.

Cerebral venous congestion.—The symptoms of passive congestion are most marked when the congestion is sudden in onset. Mechanical congestion is caused by the head-down position, by anything which impedes the flow of blood in the jugular veins, such as tight clothing round the neck, and by any cause which hinders the entry of venous blood into the thorax or heart.

Experimentally it has been found that a marked rise of cerebral venous pressure occurs when a band is tightened round the neck of a dog so as to exclude the trachea. Even flexion of the neck or contraction of the neck muscles will raise the cerebral venous pressure.

The garrotter produces loss of consciousness in his victim by suddenly occluding both the carotid arteries and the jugular veins. Cough or holding of the breath during great muscular efforts are means by which cerebral congestion is produced in ordinary life. Sir R. Gowers quotes the case of a man who exhibited clonic spasms at the climax of long coughing fits. Congestive headache is often produced by coughing. Since the cerebral capillary pressure is much increased by any great rise of intra-thoracic pressure, we might expect to find miliary aneurysms in men who have followed laborious trades. Hard toil, however, does not appear to be a sufficient cause in itself; the wall of the vessel must be predisposed, by some toxic agency, to degenerate.

It is easy to understand why cerebral hæmorrhage should most frequently occur when the veins are congested by straining at stool, muscular effort, violent cough, stooping to button boots, or during copulation; or again during sleep when the body is in the horizontal posture. The fact must never be lost sight of that cerebral capillary pressure varies absolutely with general venous, but only proportionately with arterial pressure.

Persistent mechanical congestion of the brain causes somnolence, mental inactivity, vertigo, and other symptoms which are characteristic of cerebral anæmia. Anæmia of the brain produces the same effect, whether the flow of arterial blood be reduced by a fall of arterial pressure or by a rise of venous pressure. In both cases the brain is congested with venous at the expense of arterial blood.

Cerebral venous congestion is produced during epileptic convulsions. In the status epilepticus this congestion must be greatly prolonged, and perhaps leads to those signs of meningitis which are sometimes found after death. It is important to remember that, owing to the impeded circulation and back pressure, the heart becomes engorged in diastole during the fits. The lungs become congested for the same reason. Diastolic engorgement leads to a deficient coronary circulation, for the blood circulates in the coronary arteries in inverse ratio to the pressure within the cavities of the heart; hence, perhaps, the fatty degeneration of the heart which, according to Mott, is generally found in these cases.

Although sudden anæmia of the brain excites epileptic spasms, yet we have no right to ascribe the causation of idiopathic epilepsy to cerebral anæmia. Sphygmometer readings show that there is nothing abnormal in the arterial pressure of epileptics. The fits cannot well be occasioned by vaso-motor spasm of cerebral vessels, for although these vessels possess nerves, they cannot be made to contract by any experimental means. We must fall back on the hypothesis of explosive discharge of unstable nerve elements, and suppose that in the epileptic the insulation between the dendrites of the association nerve-cells is weakened. Ramon y Cajal ascribes to the glia cells the function of an insulating material. He supposes the processes of these cells, when expanded, may act as an insulating material. Given the explosive condition of the cells, it is quite conceivable that the fits may be sometimes discharged by changes in the circulation.

In those young children in whom convulsions are excited by paroxysms of coughing, or exhaustive diarrhœa, venous congestion and anæmia of the brain are probably the immediate cause of the nerve storm.

To sum up then:—

1. *The symptoms of acute cerebral anæmia may be produced in man by*:— (i.) Embolism of cerebral arteries. (ii.) Meningeal or cerebral hæmorrhage; in these cases the anæmia of the brain may be local or general. (iii.) Failure of the heart's action. (iv.) Obstruction of the respiration; the symptoms of asphyxia being those of acute cerebral anæmia. (v.) The introduction into the system of some substance by which the blood is rendered useless for tissue respiration. (vi.) Great loss of blood. (vii.) Determination of blood to the abdomen by sudden vaso-motor paralysis. (viii.) Acute cerebral venous congestion. (ix.) Compression of the cerebral arteries.

As there is no evidence of the action of cerebral vaso-motor nerves, cerebral anæmia cannot be ascribed to spasm of the cerebral arteries.

2. *The usual symptoms of acute cerebral anæmia* are in sequence:— (*a*) Loss of consciousness; (*b*) epileptic spasm; (*c*) dilated pupils and nystagmus; (*d*) respiratory spasm; (*e*) slow heart; (*f*) rise of arterial pressure; (*g*) fall of arterial pressure, acceleration of the heart, cessation of respiration. The cortical and bulbar centres are first excited and then paralysed. The fatal symptoms arise when the bulbar circulation ceases.

3. If an animal be in a state of collapse or deep anæsthesia, or if the anæmia be slow in onset, the excitatory symptoms fail to appear.

4. In death from acute cerebral anæmia respiratory paralysis usually precedes vaso-motor paralysis.

5. Cheyne-Stokes respiration and Traube-Hering curves are common in states of partial anæmia of the bulbar centres.

6. Ligation of both carotids and both vertebrals does not entirely cut off the brain from blood. The anastomoses with the anterior spinal artery still remain open.

7. A condition of idiocy, accompanied by motor paralysis and anæsthesia, can be induced by ligation of the cerebral arteries. In this condition the cortical cells lose their stichochrome granules, and stain with methylene blue diffusely. The nuclei become swollen. If the cerebral circulation, by the opening of anastomotic pathways, become once more efficient, the condition of idiocy passes off as the cells regain their normal standard.

8. The cerebral arteries can be tied with safety if sufficient time be allowed to elapse between the successive applications of the ligatures. In ligaturing the carotid arteries in man the vessels should be obliterated slowly by means of a screw clamp, so as to allow time for the anastomotic pathways to be opened up.

9. Chronic cerebral anæmia may be caused by (*a*) loss of vaso-motor tone and a feeble heart; (*b*) venous congestion arising from valvular disease of the heart; (*c*) impeded pulmonary circulation; (*d*) pressure on the cerebral veins.

10. A slight degree of anæmia augments the excitability of the cortex. Severe anæmia reduces the cortical excitability, while it very greatly increases that of the bulbar centres. Total anæmia of the cerebrum produces almost instantaneous loss of consciousness, and abolition of cortical excitability.

The experimental effects of compression of the brain, or expression of the cerebral blood.—Boerhaave tells of a man at Paris who, at times, would beg money in a piece of his own skull, his brain being covered only with dura mater. "Upon gently pressing the dura mater he suddenly perceived, as it were, a thousand sparks before his eyes, and upon pressing a little more forcibly his eyes lost all their sight; by pressing the hand still stronger on the dura mater he fell down in a deep sleep, which was attended with all the symptoms of a slight apoplexy merely by this pressure with the hand, which was no sooner removed but he as gradually recovered from the symptoms as they were brought on, the apoplectic symptoms first vanishing, then the lethargy, and lastly the blindness; all his senses recovering their former perfection."

La Peyronie has left records of an equally interesting patient. By injecting the sac of an abscess over the region of the corpus callosum the man could be thrown into stupor or awakened, at will.

By pressing on the sac of a meningocele children can be put to sleep and the respiration slowed. Every experimenter on the subject of compression of the brain has produced in animals the same series of symptoms:—(i.) Pain, probably due to the tension of the dura mater. (ii.) Clonic spasms and circus movements when a high pressure is suddenly applied. (iii.) Constriction of the pupils, followed by dilatation: the compressed side is affected first; nystagmus may occur. (iv.) Stupor and coma. (v.) Slow heart and slow deep snoring respiration, followed, in the later stage, by respiratory gasps and acceleration of the heart. (vi.) Vomiting, emptying of the bladder and rectum.

Compression can be experimentally produced by:—(*a*) The injection of a simple fluid, such as serum or saline, into the subdural space through a tube which has been screwed into the skull. (*b*) By the production of hæmorrhage in the cranium: the carotid artery can be connected with a tube which has been screwed into the skull, and the blood can then be permitted to flow at arterial pressure into the cranial cavity. (*c*) By the local introduction of a foreign body of given limited volume: to effect this, an indiarubber bag can be inserted into the subdural space, and by means of a graduated syringe this bag can be distended to a definite volume.

An important distinction must be drawn between compression by a foreign body of limited volume and compression by the continuous injection of fluid at a constant pressure. In the latter case, if the fluid spread with ease to all parts of the cranial cavity, the pressure throughout this cavity will rise to that of the injection, and all the veins and capillaries become compressed; finally, when the injection pressure is made equal to the arterial pressure they will be obliterated. Thus, when the pressure of the injection fluid is raised gradually, fatal symptoms do not originate until the injection, and consequently the intracranial pressure, reach almost to the height of the arterial pressure. If, on the other hand, the injection fluid should not spread easily to all parts of the cranio-vertebral cavity the distribution of pressure will be various. Fluid can leak away from the cranial cavity with great ease. If there be considerable resistances to the spread of the injected fluid, and this fluid leak from the cranial cavity, it is clear that the pressure will be greater at the seat of injection than at more distant parts. If the fluid do not spread at all, but form a local collection at the seat of injection, the condition then becomes one of local compression; that is to say, the blood will be driven from the part of the brain which is compressed until the local, cerebral, vascular pressure rises to that of the injected fluid.

When compression is applied locally the brain does not transmit pressure equally in all directions. This conclusion has been reached by measurements of intracranial pressure in the cerebral, cerebellar, and vertebral chambers respectively. The brain can by no means be regarded as a bag of fluid enclosed in a rigid box; it is rather a viscous, inert mass of the consistency not of a bag of water but of a lump of putty. The brain substance itself is indeed incompressible, but the blood-vessels form an expressible sponge-work, lying chiefly on the outside of the brain mass. Each cerebral hemisphere lies in a separate chamber, partially protected from any increase of pressure in the opposite hemisphere by the strong falciform ligament. The cerebellum and spinal bulb lie in a chamber protected from cerebral pressure by the tentorium cerebelli; the narrow isthmus tentorii cerebelli is accurately filled by the peduncles of the great brain. The cerebellar chamber is connected with the vertebral canal by the narrow foramen magnum. The orifice of this foramen is blocked by the cerebellum and the spinal bulb. When fluid is rapidly injected under high pressure into the subdural space in the parietal region, the great brain is driven down and blocks the isthmus tentorii cerebelli. At the same time the cerebellum and bulb descend and completely block the foramen magnum.

When fluid is injected at low pressures the result is different, for the fluid, following the course of the cerebral vessels, can escape into the vertebral canal at each cerebral pulsation. It is necessary to bear in mind that, in all cases of simple injections, the fluid rapidly escapes from the cranio-vertebral cavity by way of the veins. Thus, to maintain pressure within the cavity, the injection must be continuous. The cause of the major symptoms of apoplexy is the arrest of blood-flow in the bulbar centres. To produce the anæmia of these centres any pressure, above the capillary pressure, applied locally to the centres is sufficient. The resistance in the bulbar capillaries need only be increased until that point is reached when the blood will flow through other pathways in the remaining parts of the brain offering less resistance. Now the bulb is so situated that the pressure of fluid injected into the meningeal space cannot be locally applied to the bulbar centres. For example, if fluid be injected through the occipito-atlantal membrane, it will not only spread to all parts of the vertebral canal, but cause the base of the brain to float up. Thus the pressure in the cranio-vertebral cavity rises everywhere to much the same point, and the injection pressure must be raised almost to the arterial pressure before the major symptoms of apoplexy arise. Local compression can, however, be applied directly to the bulbar centres by the introduction of a foreign body into the fourth ventricle; it can be applied indirectly through the descent of the brain mass when a local pressure of sufficient height is applied to the cerebrum or cerebellum. By translation of the brain mass the bulb can be pressed against the bony margin of the foramen magnum, and the capillaries of the bulbar centres obliterated.

In the case of a hæmorrhage into the cranial cavity blood can form a local collection in the brain substance, in the ventricles of the brain, between the dura and the bony wall of the skull, or in the subdural space. In some cases, on the other hand, the blood can spread like an injection fluid to all parts of the cranio-vertebral cavity. In this last case the amount of hæmorrhage, and consequent expression of blood from the brain, can reach very large limits before the bulbar centres are rendered completely anæmic. As blood clots within the cranium, and forms a foreign body, it cannot leak away through the veins in the same way as simple injection fluid, such as normal saline or serum. If the hæmorrhage and consequent clot be large, no remission of the symptoms occurs after the cessation of the hæmorrhage; but the remission can be attained immediately by freely opening the skull and clearing out the clot. It is very important to note that the mere opening of a small trephine hole is not sufficient to relieve compression; the clot still remains within, and the brain matter presses up against the trephine hole, and closes it up as a valve. To remove the compression entirely the clot must either be cleared out, or the hole made large enough to allow a compensatory expansion of the volume of the brain.

It cannot be too forcibly urged that it is not the rise of intracranial pressure but the cerebral anæmia which produces the symptoms of apoplexy. The blood-clot as a foreign body occupies a certain volume of the cranial cavity, and the cerebral blood volume is decreased to a corresponding amount. In the case of the insertion of a foreign body the pressure is raised to arterial pressure in that part of the brain where the capillaries and veins are entirely obliterated; in the neighbouring part of the brain, where the vessels are compressed but not obliterated, the pressure will be less; and in the most distant parts, which lie in some other chamber of the cranio-vertebral cavity, the pressure may remain normal. Since the major symptoms of apoplexy will not arise until the bulbar capillaries are obliterated, it follows that fatal results will ensue from a small hæmorrhage into the cerebellar chamber which in the cerebral chamber would produce but slight symptoms. A very small local hæmorrhage in the neighbourhood of the bulbar centres will suffice to obliterate the bulbar capillaries and produce death. To produce death by bulbar anæmia, in the dog, a foreign body must be introduced within the cerebral chamber = 4 to 6 c.c. in volume; in the cerebellar chamber = 1 to 2 c.c., and in the fourth ventricle = 0·5 to 1 c.c.

Since at any pressure above cerebral venous pressure the cerebro-spinal fluid rapidly leaks away from the cranial cavity, this fluid can be entirely neglected in dealing with the pathology of compression. It is absolutely erroneous to teach that increased cerebral pressure is transmitted to all parts of the cranio-vertebral cavity by the cerebro-spinal fluid. It is equally erroneous to teach that a foreign body within the cranium exerts pressure. Intracranial pressure is always circulatory in origin; the capillaries and veins are compressed, and the passage of blood blocked by the presence of the foreign body. The pressure in the compressed area is raised to arterial pressure so soon as the blood can no longer find an exit through the arterioles. When a local area of capillaries is thus blocked by a foreign body the blood takes the pathway of least resistance through the other vascular areas of the brain. In and around the compressed area, if it be extensive, there lie a large number of arterioles in which the mean arterial pressure is attained, for the outlet from these vessels is blocked. The arterial pressure transmitted from this area through the brain substance will tend to produce compression of other capillary and venous areas. At the same time, exudation of fluid at arterial pressure may take place from the blocked vessels and produce œdema of the brain, which in its turn will tend to compress neighbouring capillary areas. It follows from this that the higher the arterial pressure the more the primary area of compression may tend to spread. If this conclusion be sound, it is obvious that, in cases of cerebral hæmorrhage, the arterial pressure should be kept at a low standard, until the blocked vessels are filled with blood-clot and phagocytosis has begun to remove these clots.

When a blood-clot is removed by the surgeon inflammatory œdema of the brain may arise secondarily. The increase of vascular tension in the inflamed area of dilated arterioles will tend to compress neighbouring cerebral areas in which the capillary tension is less. This inflammatory œdema is the origin of hernia cerebri; the hernia is nature's method of relieving the cerebral compression, and indicates the line which surgical treatment should follow.

The growth of a tumour within the cranium may produce compression of the cerebral capillaries. Owing to their greater width the pressure in the capillaries of a glioma is higher than cerebral capillary pressure. The cranial contents cannot be increased, and if the quantitative ratio of cell tissue to blood-vessel be altered it must be at the expense of the blood volume. Room for the new growth can be found only by compensatory expression of blood, or by atrophy. If the tumour grow slowly, brain atrophy may take place as fast as new growth increases; thus intracranial tension may not be raised. Tumour can produce acute attacks of compression by exciting inflammation with hæmorrhages, œdema of the brain substance, thrombosis of the blood-vessels, and thickening of the meninges. As inflammatory products collect in the cranial cavity the blood volume is proportionally diminished. Whenever an inflamed area becomes hyperæmic a corresponding area must become anæmic. Thus compression of cerebral areas may be produced by meningitis and cerebral abscess.

In the choroid plexuses the resistance to blood-flow is probably less, and the capillary pressure higher than elsewhere in the brain. It is highly probable that much of the cerebro-spinal fluid is secreted from these vascular fringes and, passing into the general meningeal space, is absorbed by the veins. Should the Sylvian aqueduct or the foramen of Majendie be blocked, ventricular hydrocephalus may result, owing to the fact that the secretion takes place faster than absorption, and is at a higher pressure than that of the cerebral veins. If the veins of Galen are blocked the pressure in the choroidal fringes will rise almost to that of the arteries owing to the deficiency of anastomosis with these veins. In such case secretion becomes faster than absorption, and hydrocephalus results. A local collection of fluid in the ventricles, at a pressure higher than cerebral capillary pressure, must inevitably lead to cerebral anæmia and atrophy. Symptoms of compression rapidly appear in the adult; in the child, atrophy of brain substance and expansion of the cranial cavity. A large number of cases of hydrocephalus have now been recorded which show that the cause of the mischief is the blocking of the aqueduct or the veins of Galen by inflammatory exudations or tumours. Ventricular hydrocephalus should be relieved by draining the cavity into the general meningeal space. Dr. A. Sutherland and Mr. Watson Cheyne have recently demonstrated the truth of this conclusion: in a case of acute hydrocephalus Mr. Cheyne inserted a catgut drain leading from the lateral ventricle to the subdural space, and closed the cranial opening; under this treatment the head rapidly diminished in size and the symptoms disappeared.

From the final section of this article the following conclusions may be drawn: —(i.) The brain does not transmit pressure equally in all directions. (ii.) In regard to compression by foreign bodies there is a large amount of pressure discontinuity between the cerebral and cerebellar chambers. There is complete pressure discontinuity between the cranial and vertebral cavities. (iii.) The cerebro-spinal fluid cannot transmit a local rise of pressure to other parts of the brain. (iv.) Any pathological increase of cerebral pressure is circulatory in origin: a foreign body obliterates veins and capillaries, and thus at the seat of obliteration the cerebral presssure is raised from capillary to arterial pressure. (v.) It is not mechanical pressure but the cessation of blood-flow which produces the symptoms of compression. (vi.) The major symptoms of compression are produced by anæmia of the spinal bulb, and are exactly comparable to the symptoms of acute cerebral anæmia otherwise produced. (vii.) A far smaller foreign body kills in the bulbar region, and in the cerebellar chamber, than in the cerebral chamber. (viii.) A trephine hole does not necessarily relieve compression. The foreign body must be removed, or the opening be large enough to allow an equivalent expansion of the cranial contents. (ix.) A secondary increase of compression can be brought about by congestive or inflammatory œdema, and this condition is enhanced by high arterial pressure.

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THE REGIONAL DIAGNOSIS OF CEREBRAL DISEASE[1]

Introductory.—The regional diagnosis of cerebral disease is, in some instances, comparatively easy; in others exceedingly difficult, or not infrequently altogether impossible. The case is simplest when a stationary lesion, such as softening, invades centres, tracts, or structures, the function of which has been determined with some accuracy, whether by anatomical, physiological, or clinical research, singly or conjointly. The case is most difficult when the lesion is of such a character as to exercise a widespread disturbance in the whole intracranial structures; or when it is seated in a region the exact function of which has not been accurately determined by any method of investigation; or again when it may be compensated by the corresponding region on the opposite side, or by other parts in the same hemisphere.

The data which enable us, so far as it is at present possible, to arrive at a correct regional diagnosis are seldom anatomical, physiological, or clinical alone, but rather a combination of all three; and in the following pages, therefore, reference will be made to whatsoever method of investigation seems best calculated to throw light on the questions under consideration.

Beyond the fact, familiar since the time of Aretæus, that in hemiplegia the lesion is in the opposite hemisphere, comparatively little had been positively established by clinical investigation alone until recent years. Under conditions apparently similar there was so much diversity in symptomatology, that clinical research had not succeeded in establishing much beyond a few empirical generalisations—such as that lesion of Broca's region is the cause of certain forms of aphasia; and that irritation of the Rolandic area is the cause of unilateral convulsions, appropriately named "Jacksonian epilepsy"—generalisations extremely valuable in a diagnostic point of view, but unintelligible according to previous conceptions of the constitution of the organ of mind.

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[1] In the preparation of this article I gratefully acknowledge much valuable help from my Demonstrator, Dr. W. Aldren Turner, and from my former clinical assistant, Dr. Ashley Mackintosh, Assistant Physician, Royal Infirmary, Aberdeen.

The chaotic darkness of clinical medicine was first illumined, and a new era inaugurated, by the experimental methods first begun by Fritsch and Hitzig, and since assiduously pursued by many investigators. These observers, apart from what they have positively established, and irrespective of those individual differences and discrepancies which not unnaturally arise in the investigation of vital problems of great complexity, have so stimulated thoroughness in clinical investigation and accuracy in pathological records, microscopical and macroscopical, that they have revolutionised cerebral physiology and pathology. It is, therefore, only in the observations of the last twenty odd years that the materials are to be found on which we may base reasonable conclusions or speculations as to the probable nature and extent of intracranial lesions or disturbances of function. The observations of previous generations derive a dignity and importance more as examples of practical rules or of laws otherwise arrived at, than as contributions to establish these laws. It is unnecessary here to describe the methods which have proved so fertile in the hands of Fritsch and Hitzig and their followers, as these are now matters of general knowledge. Suffice it to say that they combine both the excitation and destruction methods; that is, excitation by electrical stimulation, and destruction by methods calculated, as far as possible, to obviate primary or secondary implication of other structures, near or remote. Excitation and abolition, or impairment, of function practically embrace all the phenomena which can be induced by cerebral lesions; all lesions may therefore be classified into two great groups, *irritative* and *destructive*. These two classes of lesions, however, are not separable by hard and fast lines; for a lesion which is irritative in the first instance may in time prove destructive, and cause annihilation of the function which formerly it excited; and, similarly, lesions, essentially destructive from the first, may be the centre of a zone of irritation, so that irritative phenomena, of greater or less range, are superadded to the paralytic symptoms. An effect intermediate between that of irritation and destruction must also be taken into account, namely, the perturbation, inhibition, or temporary cessation of the functions of parts more or less remote from the seat of actual lesion. This is a dynamical influence most frequently seen in connection with sudden lesions, and probably due to the rupture of the solidarity which normally exists between the several intracranial centres; but whether it should be regarded as being irritative or destructive, is not quite clear. As, however, the ultimate effect is the abolition or impairment of function, it may rightly be classed among the destructive lesions, and designated indirect, in contradistinction to those which act directly on the parts in which they are primarily situated. It is the lesions of this last class which create so much difficulty in the accurate regional diagnosis of cerebral lesions, and which also largely explain the discrepancies which abound in the reports of experimental lesions in the brains of the lower animals; for the estimation of the duration and range of these inhibitory phenomena is by no means easy to calculate. It is necessary, therefore, to bear these considerations carefully in mind in attempting, among the phenomena of cerebral disease, to separate the essential from the accidental, and to refer to their respective causes the various symptoms met with in connection with any given lesion.

Destructive lesions are exemplified in their simplest form in atrophic degenerations; necrobiotic softenings, conditioned by non-irritative vascular occlusion; and slight aseptic traumatism.

Irritative lesions are exemplified in inflammatory processes, neoplasms, and septic foci; while *indirect or inhibitory lesions* are exemplified in pathological processes of a sudden character, such as extensive injury, hæmorrhagic extravasation, and the pressure and other remote effects of intracranial tumours.

For the purposes of this chapter the morbid anatomy of intracranial lesions is unimportant and will not be considered. What we have to consider *quâ* regional diagnosis, is whether the lesion be essentially irritative or destructive in character; and whether it be such as is calculated to exercise a disturbing influence on regions anatomically remote from the part actually invaded. A knowledge is also assumed of the general features of cerebral anatomy, and of the convolutional arrangement of the cortex, as well as of the vascular supply.

LESIONS OF THE FRONTAL LOBE.—The frontal lobe, as here defined, is not coextensive with the frontal lobe of anatomists, but is restricted to that portion of the cerebral hemisphere which lies anterior to the precentral sulcus and its ideal prolongation upwards to the longitudinal fissure. This line cuts off the base of the superior frontal convolution which functionally should be included in the Rolandic area. Physiological experiment further indicates that the frontal lobe as so defined must be divided into a *prefrontal* and a *post-frontal* area. The division between the two is indicated in the brain of the macaque monkey by a shallow transverse sulcus, the homology of which in man is somewhat doubtful.

Electrical irritation of the posterior half, or post-frontal area, causes movements of the head and eyes to the opposite side; while irritation of the prefrontal area is uniformly negative, or is occasionally accompanied by ocular movements only. It would appear, however, from Russell's experiments that the lateral, or conjugate, deviation of the eyes to the opposite side becomes modified, to upward or other movements, when the external rectus of the opposite eye is divided—a fact which is taken to indicate that other ocular movements are represented in this area, but are usually concealed by the stronger and more easily excited lateral movements.

Experimental lesions of the frontal lobe, according to my earlier observations, lead to a tract of descending degeneration which occupies the lowest and innermost area of frontal sections of the internal capsule, and the most mesially situated fibres of the crus. This degeneration I had not been able to trace beyond the upper part of the pons. More recent experiments in conjunction with my colleague Dr. Turner confirm the existence of this tract of degeneration after lesions of the frontal lobe. Its termination, however, is still uncertain. Flechsig is of opinion that it connects the frontal lobe with the cerebellar hemisphere,

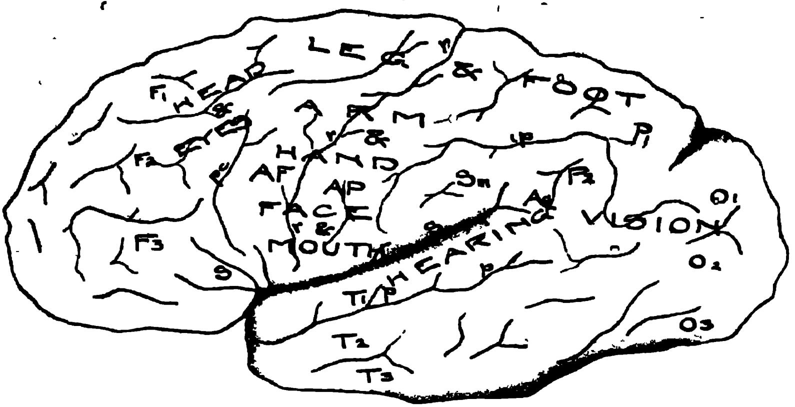

FIG. 20.—Convex aspect of cerebral hemisphere. F1, F2, F3, Superior, middle and inferior frontal convolutions; AF, ascending frontal, or precentral convolution; AP, ascending parietal, or postcentral convolution; P1, P2, superior and inferior parietal lobule; Sm, supramarginal lobule; Ag, angular gyrus; O1, O2, O3, superior, middle and inferior occipital convolutions; T1, T2, T3, superior middle and inferior temporal convolutions; *pc*, precentral sulcus; *r*, fissure of Rolando; *s*, fissure of Sylvius; *ip*, intraparietal sulcus; *p*, parallel fissure.

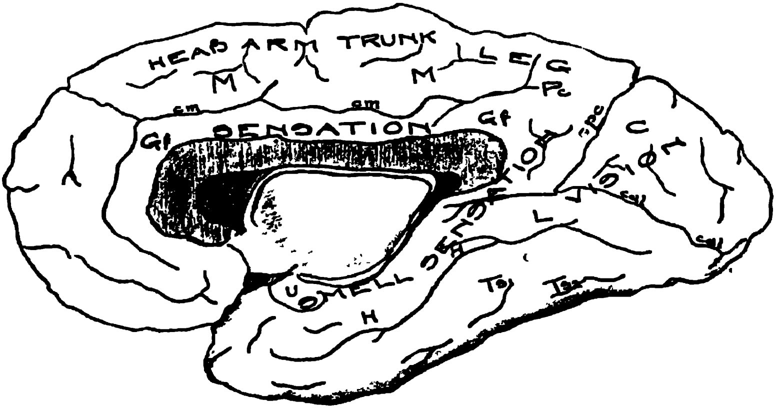

FIG. 21.—Mesial aspect of cerebral hemisphere. M, Marginal gyrus; Gp, gyrus fornicatus; H, hippocampal gyrus; U, uncus; Pc, precuneus; C, cuneus; L, lingual lobule; Ts, fusiform lobule; *cm*, calloso-marginal fissure; *po*, internal parieto-occipital fissure; *cal*, calcarine fissure.

indirectly through the gray matter of the pons. Similar degeneration has been described by Brissaud as the result of lesions of the anterior limb of the internal capsule in man. As the degeneration could not be traced into the anterior pyramid, he concludes that the tract of the foot of the crus connects the frontal region with the motor nuclei of the medulla oblongata.

Bechterew and Witkowsky have observed atrophy of the cells of the substantia nigra after old lesion of the anterior part of the internal capsule with descending degeneration in the crus. This Turner and I have confirmed in some recent experiments on the prefrontal lobe. The frontal lobe is considered by Bechterew to be connected with the occipital lobe by a large tract of fibres, termed the fasciculus longitudinalis superior. Removal of the prefrontal lobe, however, does not, according to our observations, occasion any degeneration traceable into the occipital lobe.

When the post-frontal region in monkeys is destroyed, a temporary deviation of the head and eyes to the side of the lesion always occurs. This, however, is only transient, even when the destruction has been almost, if not absolutely complete. In two experiments which I have described (10) the post-frontal area was destroyed in both hemispheres. The animals were unable to turn the head and eyes to either side for a day after the operation. At first they were unable to look around when sounds were made in close proximity to the ear, or, if they did, they moved the trunk and head "en masse."

Removal of the prefrontal region alone causes no discoverable physiological symptoms, either sensory or motor. I found, however, in several instances, that, after the symptoms which followed destruction of the post-frontal area had entirely disappeared, the subsequent destruction of the prefrontal area induced paralysis of the head and eyes of exactly the same nature as before. Thus in one case, after cauterisation of the whole of the excitable area on its convex as well as mesial aspect, the animal, which at first exhibited marked conjugate deviation of the head and eyes to the side of the lesion, recovered within three days to such an extent that the defects were no longer perceptible. Extirpation of the prefrontal region anterior to the former lesion, a month later, reproduced the conjugate deviation of the head and eyes to the side of the lesion. The conjugate deviation of the eyes continued for some time after the distortion of the head had been recovered from, but within three days this defect had also disappeared.

It is difficult to remove both frontal lobes thoroughly without injuring the head of the nucleus caudatus, or causing lesions incompatible with long survival. However, in a recent experiment we have succeeded in removing the whole of both frontal lobes after an interval of more than a month between the two operations. The removal of each lobe was followed by temporary conjugate deviation of the head and eyes; but, notwithstanding the apparent total removal of the frontal lobe on both sides, there was no permanent inability to move the head and eyes freely in all directions. It would appear, therefore, from this experiment that, though the frontal lobes are evidently in relation with the movements of the head and eyes, complete destruction of these regions does not give rise to permanent paralysis; and still more striking is the fact that in this same animal, after an interval of five weeks, destruction of the angular gyri,—in which, as we shall see hereafter, some are inclined to place the centres for the ocular movements,—did not cause ptosis, or paralyse the ocular movements. The animal, however, speedily succumbed to the shock of the operation, so that our period of observation was unfortunately short.

When the lesion is accurately confined to the frontal lobe, and does not extend backwards to the motor region proper, temporary conjugate deviation of the head and eyes to the side of lesion is the only discoverable physiological symptom.

Hitzig (14), however, after destruction of the frontal lobe, has observed, along with impairment of the movements of the limbs, affection of the vision of the opposite eye, and is inclined to think that this is not accidental, but that the frontal lobe is related to vision. In the last-mentioned experiment, we observed similar effects after complete extirpation of the left frontal lobe, together with a portion of the base of the superior frontal, anterior part of the gyrus fornicatus, and a part of the head of the nucleus caudatus. The lesion was therefore not strictly confined to the frontal lobe. In addition to the conjugate deviation of the head and eyes there occurred a paretic condition of the right arm and leg (continuing longer in the leg), and also a slight and temporary impairment of tactile sensibility, as well as distinct right hemianopsy. These symptoms, except the weakness in the right leg, entirely disappeared in the course of a month; and, as will be shown by other experiments, cannot be attributed to lesion of the frontal lobe, as such.

The recorded cases in man of injury and disease strictly confined to the frontal lobes accord with the negative character of experimental lesions in monkeys, so far as relates to the sensory and motor faculties. The majority may also be described as latent, that is, not revealed by any strictly localising symptoms. Of seventy-three cases of lesion of the frontal lobes, unilateral and bilateral, which I have been able to collect from various sources, twenty-four were traumatic, caused, that is, by fractures, gunshot wounds, or perforating instruments; four were due to hæmorrhage; twelve to abscess, mostly secondary; in one the abscess was secondary to mastoid disease; twenty-five were due to tumours; and one was a case of atrophy. The remainder were tabulated as "softening."

Hæmorrhage.—With the exception of the inferior and posterior part of the middle frontal convolution, the frontal lobe, both on its convex and mesial aspect, is supplied by the anterior cerebral artery. This artery does not appear to be particularly liable to rupture or occlusion; hence hæmorrhages, apart from traumatism, and embolic or thrombotic softening are comparatively rare.

Traumatic lesions.—Many cases are on record of recovery, without any obvious impairment of sensation or motion, after traumatic injuries of a most extensive character. One of the most remarkable is the famous American crowbar case. The subject of the lesion was a young man aged twenty-five. While engaged in stamping a blasting charge in a rock with a pointed iron bar, 3 ft. 7 in. in length, 1¼ in diameter, and weighing 13½ lbs., the charge suddenly exploded. The bar, propelled with its point first, entered at the left angle of the patient's jaw, and emerged anterior to the coronal suture. The patient was only momentarily stunned, and recovered without any defect of sensation or motion, except the ptosis and loss of vision in the left eye, due to local injury of the optic and oculo-motor nerves. Examination of the topographical relations of the brain and skull clearly proves that the track of the instrument was anterior to the Rolandic or motor area, and could only have injured the excitable post-frontal area, unilateral lesions of which, as we know from experiments, cause very transient symptoms only. Whether they occurred at all in this patient has not been noted.

Another similar and not less remarkable case has recently come under my observation. It is that of a young man, aged twenty-six, subject to epileptic fits, who, apparently in a dazed condition, and without purpose, discharged a pistol through the roof of his mouth. The bullet lodged under the frontal bone, which it fractured and partially raised. The fractured bone was elevated and the bullet extracted by Dr. Walker of Peterborough. The position of the opening was half an inch to the left of the middle line, and an inch anterior to the coronal suture. The patient entirely recovered, with the exception of ptosis and weakness of the superior rectus in the left eye, and diplopia for objects situated above the horizontal line. When I saw the patient, within three months of the injury, there was no trace of any affection of motion or sensation, general or special.

Many other cases of unilateral traumatic lesion are on record; but it is not necessary to refer to them in detail, as they merely illustrate the same negative character of such lesions.

Of bilateral lesions one of the most interesting is that reported by Davidson. This was the case of a labourer who received a blow on the side of the head with an iron hook, which fractured the frontal bone and injured the brain as far back as the coronal suture. The injured portion of the brain included, on the right side, the greater part of the superior and middle frontal convolutions; and, on the left, a considerable amount of the middle of the superior frontal convolution. With the exception of an occasional spasmodic extension of the right arm, there were no other symptoms in the domain of motion or sensation.

Abscesses in the frontal lobe are, for the most part, consecutive to traumatic injuries or perforating wounds, or to diseases of the frontal, ethmoid, or orbital bones; but a case has been reported by Prentiss of abscess in the left anterior lobe secondary to abscess in the right petrous bone and lateral sinus. In most of these cases there was entire absence of sensory or motor symptoms, and in many there was nothing noteworthy, or recorded, in reference to the mental condition. In some, however, to be alluded to subsequently, the mental condition was such as to attract attention.

Pitres reports a case of injury to the left frontal region which gave rise to headache, vomiting, and death two months afterwards, without having caused any obvious affection of intelligence, or paralysis of motion or sensation. After death an abscess was found in the centre of the left frontal lobe. Macewen reports a case of a blow on the forehead by a stone; suppuration occurred in the wound, followed by headache, drowsiness, vomiting, and occasional rigors, and double optic neuritis. Apart from these general symptoms there was no affection of motion or sensation. The patient was trephined, and an abscess was evacuated in the left frontal lobe.

Tumours form a large proportion of the cases of disease of the frontal lobe, either in the substance of the lobe itself, or growing from the dura mater, and compressing and destroying the subjacent cortex. Apart from the general symptoms of cerebral tumour—namely, headache, vomiting, optic neuritis and epileptiform convulsions—in many instances there has been nothing to enable one to indicate with certainty at the seat of the disease.

Hun reports a case of one and a half year's duration, with symptoms merely of general weakness, in which death suddenly occurred in an attack of coma. A tumour the size of a small hen's egg was found in the lower part of the frontal lobe. Dr. Hale White reports a case of a woman who for one month had suffered from headache, vomiting, and occasional loss of sight. Optic neuritis existed, but there were no special symptoms. The patient was found one day dead in bed. The post-mortem examination revealed a glioma in the frontal lobe. Other similar cases might be mentioned.

In some cases the tumour has been indicated not by any symptoms referable to the frontal lobe, as such, but by affection of the nerves in the anterior fossa. Thus, Schlager, quoted by Lepine, reports a case of a woman who for some years before her death suffered from vertiginous sensations and epileptiform attacks, preceded by an olfactory aura. There was no other affection of motion or sensation. She died comatose. A carcinomatous tumour, the size of a pigeon's egg, was found in the substance of the left frontal lobe, which had completely destroyed the left olfactory tract.

In other cases there have been observed indications of paresis or paralysis of the ocular nerves, fulness or protrusion of the eyeball, and failure of vision in the eye on the side of lesion, not to be accounted for by the general optic neuritis. These observations are true also of abscesses.

Griffith and Sheldon have reported a case of tumour occupying the anterior fossæ, and thrusting itself between the frontal lobes, chiefly destroying the orbital surface and exerting pressure upon the olfactory tracts and optic nerves. The symptoms in this case, in addition to those of cerebral tumour in general, were impairment of memory, loss of smell on the right side, and paresis of the left abducens. Before death both discs became atrophied, with almost total loss of vision. There were no motor or sensory symptoms, and, except for some attacks of maniacal excitement, the mind was quite clear and coherent until shortly before death.

In another group of cases there has been superadded to the general symptoms of cerebral tumour some affection of motion and sensation on the opposite side of the body. This is true not only of tumours, but also of abscesses, and of inflammatory softening extending backwards from the primary lesion of the frontal lobes. These symptoms are undoubtedly due to pressure on, or direct implication of, the Rolandic zone or the hemisphere in general, and often afford valuable diagnostic indications of the seat of the disease, which in its earlier stages may not have declared itself by any definite symptoms. Thus in a case, recorded by Treves, of fracture of the frontal bone and laceration of the brain, at first there were no motor or sensory disturbances; but, as encephalitis advanced, convulsions and paralysis, limited to the left arm, set in, and the patient died on the seventh day after the accident. After death the anterior two-thirds of the convolutions of the right frontal lobe were found destroyed, and behind this there was a zone of congestion and softening. On the left side about one-fourth of the frontal convolutions was destroyed. The motor symptoms in this case were clearly due to the irritation and subsequent destruction of the motor centres of the Rolandic area by the inflammatory action. In a case reported by Gouget, the patient was admitted in a state of coma, with right hemiplegia and conjugate deviation of the head and eyes. Ten days before he had a sudden temporary loss of consciousness. A few days later another similar attack occurred, followed by weakness of the right side. This lasted for two days. Again he became comatose, and died. After death an abscess, containing three to four spoonfuls of pus, was found occupying the inferior part of the second left frontal gyrus. The pus cavity extended within two centimetres of the apex of the frontal lobe and as far back as the head of the nucleus caudatus, but did not communicate with the ventricle. Lannelongue and Mauclaire report the case of a child who, after a fall downstairs, exhibited palsy of the left lower limb and contracture of the left arm. There was conjugate deviation of the eyes to the left, palsy of the right lower facial region, slight paresis and anæsthesia of the right arm, and contracture and hyperæsthesia of the right leg. After death lesions were found in both frontal lobes: on the right, contusion of the frontal gyri and the middle part of the ascending frontal and inferior parietal; and, on the left, contusion at the base of the second left frontal involving slightly the third frontal gyrus. This penetrated one centimetre into the cerebral substance.

Bruns has described four cases of tumour of the frontal lobe in which, in addition to some degree of paresis or paralysis of a hemiplegic or monoplegic character on the opposite side, there were also disturbances of equilibrium and co-ordination. In one case (sarcoma affecting both frontal lobes and involving the genu of the corpus callosum) there was instability of equilibrium with a tendency to retropulsion; and in another (hæmatoma indenting the left frontal lobe) the patient could not stand without support, and had slight paralysis of the right hand and face. A case of frontal lobe tumour with purely cerebellar symptoms has been recently under my own care. This combination of symptoms, as Bruns indicates, is in all probability due to pressure exerted backwards on the cerebellum in the line of thrust, and is peculiar to tumours of the frontal region.

The symptoms hitherto described are in accordance with the negative effects, *quâ* motion and sensation, which have been uniformly found in monkeys after extirpation of the frontal lobes proper; and the occurrence of spasmodic or paralytic affections of the limbs is also in harmony with the effects of lesions primarily or secondarily involving the motor centres of the Rolandic area. It is a question, however, whether the symptoms of injury and disease of the frontal lobes in man entirely bear out those which have been described as the result of irritation and destruction of these regions in monkeys and other animals. It has already been stated that irritation of the post-frontal area uniformly causes conjugate movement of the eyes and head to the opposite side, and that extirpation of this region is uniformly followed by conjugate deviation of the head and eyes to the side of lesion. This, however, is only of temporary duration, even when the post-frontal centres have been entirely destroyed; and it would be in accordance, therefore, with the results of experimental physiology that such phenomena should not occur in man except in connection with irritative or sudden destructive lesions of the area in question.

Irritation would cause conjugate movement of the eyes to the opposite side, while sudden destruction would cause deviation to the side of lesion, owing to the non-antagonised action of the centres in the opposite hemisphere. Slowly progressive lesions, however, need not reveal themselves by conjugate deviation, as compensation would be effected as rapidly as the process of destruction. Chouppe has reported a case of tuberculous meningitis in which, in addition to the pain in the head, vomiting, and so forth, the most remarkable symptom was rotation of the head and eyes to the right. This could be overcome with moderate effort, but the head and eyes returned to their abnormal position when left to themselves. There were no symptoms of paralysis or contracture. After death a superficial lesion of the size of a franc piece was found on the superior part of the middle frontal convolution in the left hemisphere. This may be regarded as in all probability an irritative lesion of the oculo-motor area. In Gouget's case (12) there was conjugate deviation of the head and eyes to the side of lesion; but as it was accompanied by general hemiplegia, it does not admit of being referred to limited lesion of the left frontal lobe. In Lannelongue's case (17) the conjugate deviation of the head and eyes to the left side might perhaps be attributed to the lesion at the base of the second left frontal convolution.

Apart from the cases which I have quoted, however, I do not find any very satisfactory evidence of conjugate movement of the head and eyes having been observed in connection with lesions implicating the central regions homologous with the ocular centres in the monkey.

I have elsewhere (9) described the *mental symptoms* observable in monkeys after extirpation of the frontal lobes. These, however, only occur when the lesion is bilateral, and some experimenters have failed to corroborate them. They may easily escape superficial observation, and it is only by careful comparison of the animals before and after operation that they are capable of being detected, and even then it is difficult to state their nature precisely. The animals do not appear to have entirely lost any of their mental faculties. They, however, exhibit a curious listless apathy or purposeless restlessness, and certainly lose the intelligent keenness and cunning which they possessed before the operation. I have confirmed these observations in a recent experiment (1894) in conjunction with my colleague Dr. Turner. The whole of the frontal lobes, including the oculo-motor centres, were entirely extirpated on both sides. Though the animal recovered perfectly, with the exception of slight weakness in the right leg, and enjoyed excellent physical health, the mental alteration, of the character just described, was so striking that there could be no question of its reality.

It has already been stated that in a large proportion of the cases of disease of the frontal lobe in man no mental symptoms have been noted; but in others they have been so marked as to have deserved special mention in the clinical records. Many of the cases have, however, been tumours, and it is therefore a question how far these symptoms should be attributed to lesion of the frontal lobes, as such, apart from general disturbance of the brain. But mental symptoms, of the character above described, have also been observed in connection with lesions of a purely stationary character, not calculated to affect the brain as a whole. A case has been recorded by Baraduc of atrophy of the frontal convolutions in both hemispheres, due to obliteration of the arterial blood-supply. The lesions occupied the first, second, and third frontal convolutions, and also the internal aspect of the frontal lobes. The ascending frontal and parietal gyri and paracentral convolution were narrowed, but the rest of the brain was normal, except in the region of the inferior parietal lobule of the right hemisphere. The patient, who was an inmate of the Hospice de Menages for six years, exhibited no paralysis of sensation or motion. He was, however, in a state of dementia, marching about restlessly the whole day, picking up objects from the ground, never speaking, and quite oblivious of all the wants of nature. In Davidson's case, above referred to, the only symptoms of the cerebral lesion, apart from occasional spasm of the right arm, were of a psychical nature. Though the patient seemed to understand all that was said to him, and did what he was told, "every action he performed left the impression on the mind of the observer that it was purely automatic or machine-like." Marked mental deficiency has also been frequently observed in connection with arrested development or atrophy of the frontal lobes. Thus, in the case of a girl aged 15, reported by Cruveilhier, who had remained in a state of idiocy from birth, the anterior two-thirds of the frontal lobes were completely deficient.

The association of mental symptoms with tumours of the frontal lobes has been remarked in many recent cases. According to Starr, of twenty-three cases of tumour of the frontal lobes mental disturbances occurred in one-half.

I have myself recorded a case (11) of a man aged 40, who, when he came under observation, in addition to the general phenomena of cerebral tumour, exhibited symptoms essentially mental. These consisted chiefly in great failure of memory, inability to concentrate his attention, general mental hebetude, and a continual tendency to fall asleep. During the period of observation from June to January the mental symptoms remained essentially the same, but in addition there sometimes appeared to be relative weakness of the right side of the body, never amounting to actual paralysis. He died comatose after an attack of influenza. After death it was found that the anterior portion of the left frontal lobe was occupied by a large tumour, about three inches in diameter, attached to the dura mater in front, from which it had apparently grown and pressed backwards on the frontal lobe. During life the probability of tumour being situated in this region was expressed, both from the mental symptoms and signs of pain on deep pressure over what proved to be the seat of the tumour. It was impossible to arrive at any accurate conclusion with regard to the sense of smell in this case, but if the sense of smell had been certainly affected there would have been no hesitation in operating for the removal of the disease. Professor Allbutt has narrated a case to me of glioma the size of a large pigeon's egg in the left frontal lobe of a middle-aged woman. The growth of this tumour, which must have been of long duration, gave rise to no symptoms until the patient was within a few days of death. She then became dull, forgetful, torpid and sleepy, and soporose hysteria might have been diagnosed had it not been for the lack of corroborative evidence (the patient was a lady's maid, and an active and sensible person), and for the presence of highly swollen optic discs. The sopor deepened until the death of the patient, about the tenth day after the earliest symptoms and without any further diagnostic indication. There was no cranial pain, on percussion or otherwise, at any time.

A successful operation for tumour in this region was performed by Durante. The symptoms on which Durante based his diagnosis of tumour of the frontal lobe were loss of memory and change of disposition, anosmia, and some downward and outward displacement of the left eye. The patient recovered perfectly after removal of the tumour. The symptoms in a case recorded by M'Burney and Starr were headache, vomiting, and optic neuritis, and a severe and constant pain in the frontal region, increased by pressure or percussion. There was marked mental dulness and difficulty in concentration of attention, together with slight hemiplegic weakness on the right side. In this case an operation was decided on, and a capsulated tumour was removed which had involved the posterior extremity of the second frontal convolution, the adjacent portion of the first frontal and the upper half of the ascending frontal or anterior central convolution. A case with similar symptoms, dependent on an infiltrating glioma the size of a hen's egg in the left frontal lobe, is related by Knapp. For other recent cases, in addition to those specially mentioned, see Gilman Thompson, Morrison, Burr, Keen.

An examination of the cases above recorded, as well as of many others which are to be found in medical records, shows that the regional diagnosis of lesions of the frontal lobes, independently of external indications, such as fracture and the like, may be a matter of doubt if not of absolute impossibility. It is abundantly evident that extensive lesions may exist in one or even both frontal lobes without revealing themselves in any impairment of motion or sensation, general or special; or even in mental symptoms of such character as to call for special description.

Summary.—(i.) Lesions of the frontal lobe may therefore be said to be not infrequently latent.

(ii.) On the other hand, in some cases, especially if the lesion is bilateral, and even in the case of lesions which are not calculated to cause pressure or disturbance of the brain in general, there may be mental symptoms of which the chief characteristics are failure of memory, hebetude, apathetic indifference or tendency to sleep, vague restlessness, and inability to concentrate attention.

(iii.) The regional diagnosis of lesion of the frontal lobe is rendered more probable if, in addition to the psychical symptoms, there occur convulsive or paralytic symptoms, monoplegic or hemiplegic, on the opposite side of the body. These symptoms are indicative of extension of the lesion backwards into the Rolandic area.

(iv.) The diagnosis of lesion of the frontal lobe may be made with still greater certainty, if, in addition to the symptoms enumerated under paragraphs ii. and iii., there are signs of pressure in the anterior fossa; consisting in protrusion or displacement of the eyeball, with perhaps unilateral loss of vision, anosmia, and paralysis of one or other of the oculo-motor nerves.

(v.) The diagnosis is confirmed if, in addition to some or a combination of several of the above-mentioned symptoms, there is pain on deep pressure on the frontal bone. This local pain may, however, be entirely absent if the lesion be subcortical, and is especially to be found in cases of tumour causing tension or irritation of the dura mater.

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Disease of the Rolandic or Motor Region.—The Rolandic or motor region, as defined by experiments on monkeys (Ferrier (25), Schäfer, Horsley and Beevor), includes the convolutions which bound the fissure of Rolando; namely, the ascending frontal or precentral convolution and the base of the superior frontal, the ascending parietal and its broadened base, the postero-parietal or superior parietal lobule, together with the corresponding portion of the marginal convolution which forms the mesial aspect of the hemisphere. In this region are situated specialised areas, stimulation of which by the electric current gives rise to certain definite movements of the opposite side of the body; namely, of the trunk, limbs, facial, lingual, and laryngeal muscles; and destruction of which causes hemiplegia, if the entire region be destroyed; or limited or dissociated paralyses or monoplegias, if individual areas only are destroyed; the paralysis or monoplegia in this case corresponding to the movements which are excited by the electric current.

At the upper extremity of the central convolutions, and extending over the margin of the hemisphere into the posterior part of the central convolution—paracentral lobule,—electrical stimulation gives rise to movements of the *lower extremity*. The movements so excited vary with the position of the electrodes in this area. Behind the fissure of Rolando they are chiefly, or exclusively, of the foot or toes; in front of the fissure of Rolando they are combined with movements (flexion) of the leg and thigh. With minimal stimulation (Horsley and Beevor) it is possible to note differentiated movements of the lower limb with still greater minuteness, and, in particular, the great toe may be separately thrown into action by stimulation at the uppermost extremity of the fissure of Rolando.

Below the leg area, and partly in front of it, and occupying the middle third, or rather middle two-fourths, of the central convolutions, there is an area stimulation of which causes movements of the *upper* *extremity.* In this area one may distinguish, more or less completely, protraction and retraction of the upper arm, flexion and supination of the forearm, and movements of the wrist, thumb, and fingers. The proximal movements are more particularly represented in the upper part of this area; the distal movements, namely, those of the fingers and thumb, in its lower part. By minimal stimulation of the lower extremity of the intraparietal sulcus the thumb alone may be thrown into action.

Below the arm area, and occupying the lower third of the central convolutions, electrical stimulation causes movements of the *face, mouth, tongue, and larynx.* In the upper part of this area stimulation in front of the fissure of Rolando causes movements of the upper facial muscles, and behind this fissure, of the platysma myoides. In the lower portion, stimulation causes movements of the mouth and tongue—protrusion of the tongue being generally caused by stimulation anterior, and retraction by stimulation posterior to the fissure of Rolando. In the anterior division, also, of the lower half of this area stimulation gives rise to phonatory closure of the vocal cords (Horsley and Semon), and occasionally, as I have found in some animals, to actual phonation.

The Rolandic region, as has been stated, extends over the margin of the hemisphere into the marginal convolution, on the mesial aspect of the hemisphere; and stimulation of this convolution from before backwards gives rise to movements of the arm and shoulder girdle, trunk and pelvis, and lastly of the leg, foot, and toes (Horsley and Schäfer). Movements excited by stimulation of this region, however, are less capable of being exactly discriminated one from another.

Whether in respect to the effects of electrical stimulation of the cortex a parallelism exists between the lower animals and man, is a question which has been answered in the affirmative by some recent observations in patients in whom the brain has been exposed with a view to the extirpation of lesions supposed to be the cause of focal epilepsy. Cases of this kind have been reported by Mills (47), Keen (39), Lloyd and Deaver (41), Nancrede, Ransom, and Dana (16). The results have been such as to show that the same kinds of movements occur in man as in the monkey, though we cannot say that a precise anatomical homology has yet been established. The excitation method is now, indeed, employed by surgeons as a guide to the exact localisation of a centre which it is desired to extirpate.

The results of the excitation method show that whether the individual segments of a limb are separately localised, or are represented more or less throughout a common area, the areas as a whole are completely differentiated from each other. Thus no movements of the leg can be excited from irritation of the facial centre, nor of the face from the leg centre.

The fact that stimulation of the margin of a given area is apt to produce combined movements of this and the adjoining area, does not justify the conclusion that this portion subserves conjoint functions. It is more likely that the excitation method cannot accurately map out the boundaries of respective areas, for areas which are in closest proximity to each other, anatomically and functionally, are apt to be discharged by diffusion of the same stimulus. In respect to the movements of a limb, however, it is found that, though a particular movement may be frequently isolated by minimal stimulation of a defined point within the general area, yet this same movement may occur along with others when another part of the area is under stimulation. This may be interpreted either on the supposition that the particular movement is represented throughout the whole of the area, or that it is the result of a diffusion of the stimulus to the particular area previously defined by minimal stimulation.

Probably both suppositions are more or less true. Inasmuch as the movements excited from the cortex are acts, and not mere muscular contractions, it is probable, from a consideration of the results of stimulation of the roots of the brachial and crural plexuses, that the same movement may occur in different combinations; and that there may be, therefore, a multiple representation of a given muscular group. But this does not justify the conclusion that one and the same portion of gray matter can subserve different functions. My opinion is that functional differentiation depends on anatomical conditions, and that these are as distinct in the cortical centres as one organ from another. The centre for the thigh can no more function for the arm or hand than the eye for the ear, or conversely. But multiple representation will permit of the movements more especially centralised in a given area being carried out, in certain combinations at least, notwithstanding the destruction of this particular area. Hence, for instance, destruction of the thumb area proper may not entirely paralyse the thumb, if this be represented also elsewhere.

The same difficulty, which is encountered in defining the complete differentiation of movements by electrical stimulation, is observed in the case of destructive lesions which invade the boundaries of respective areas. Hence a lesion which paralyses the thumb is apt to cause a greater or less degree of paralysis of the whole arm; or, it may be, of the leg also, if the lesion extend beyond the boundary of the arm centre.

It has been established, by experiments on monkeys, that destruction of the areas excitation of which produces definite movements, causes volitional paralysis of the same movements on the opposite side of the body, which varies in extent and completeness with the destruction of the respective centres. When the destruction is complete the paralysis is permanent, and is followed by degeneration of the pyramidal tracts of the spinal cord and contracture of the paralysed limbs (23). Some movements, however, are so bilaterally represented in each cerebral hemisphere that even complete extirpation of the centres in the one hemisphere causes little or no obvious impairment of these movements; and, in order that paralysis should result, it is necessary that the centres on both sides should be destroyed. Of this character are, first, those of the trunk, hip, and shoulder girdle, which have their centres in each marginal convolution; thus in order that these movements may be paralysed it is necessary that the marginal convolution should be destroyed in both hemispheres; secondly, the movements of the mouth and tongue; and, thirdly, those of the larynx.

Unilateral extirpation of the oro-lingual or of the laryngeal centres, at the lower extremity of the Rolandic area, does not appreciably impair the movements of articulation or of phonation; whereas they become completely paralysed when the centres are destroyed in both hemispheres. It would appear that such movements as may not be primarily represented bilaterally in each hemisphere are secondarily associated, according to Broadbent's hypothesis, by commissural fibres between the respective bulbar and spinal nuclei. That bilateral representation of movements in the cerebral hemispheres is founded not merely on functional but on structural relations is proved by the fact that secondary degeneration in the pyramidal tracts occurs on both sides in accordance with the degree of representation in each hemisphere. Thus, according to the researches of Sherrington, if the lesions affect the centres for the limbs only, the pyramidal degeneration is almost entirely on the opposite side; whereas it is decidedly bilateral if the lesion invade the marginal convolution: and this is still more pronounced if the lesion invade the laryngeal centre.

These facts show that even in the case of the limbs, each hemisphere represents both sides of the body, mainly the opposite, but also to some extent the same side; and this is in accordance with the observations of Brown-Séquard, Pitres, and Friedländer—that lesions which cause hemiplegia of the opposite side cause also some diminution in the dynamic energy of the limbs on the same side.

The bilateral relations of each hemisphere, so well marked in monkeys and man, is still more pronounced in some of the lower animals; and it is this bilateral representation which accounts for a large amount of recovery, even when the motor centres of one hemisphere have been entirely destroyed. The recovery extends more particularly to those movements which are more or less habitually associated with those of the other side, and least of all to those movements which are most independent and most highly specialised. Hence, in cortical motor paralysis the arm is more paralysed than the leg, and the distal movements of the limb more than the proximal.

Cortical motor paralysis is essentially paralysis of voluntary movement, or that which implies conscious discrimination; and is consistent with the retention of co-ordinated movements of the same muscular groups of considerable complexity of the automatic or instinctive order. The degree in which these are retained, after extirpation of the motor cortex, varies much in different orders of animals; they are highest in fishes and birds, and lowest in monkey and man.

Lesions invading the Rolandic area are divisible into two great groups; first, irritative lesions, and, secondly, destructive lesions. Practically, however, they cannot be separated from each other by hard and fast lines, for not infrequently those lesions, which ultimately prove destructive of the centres which they invade, are primarily, or at some time in their course, associated with irritative phenomena.

Irritative lesions.—Irritative lesions are so called because they tend to induce a sudden or explosive discharge similar to that which may be induced artificially by long-continued electrical stimulation of the cortex. The discharge is apt to leave a condition of exhaustion, so that a paralytic condition, more or less enduring, may be observed to follow the epileptiform convulsion. This may be a post-epileptiform hemiplegia or a post-epileptiform monoplegia, according as the convulsion has been general or limited. As the pathology of unilateral epileptiform convulsions was first clearly indicated by Dr. Hughlings Jackson, the affection has been appropriately termed "Jacksonian epilepsy."

Jacksonian epilepsy may be taken in all cases to indicate the existence of some irritative lesion of the cortical motor centres. The mere occurrence of a unilateral convulsion, or of an epileptic attack in which the convulsions are exhibited mainly on one side, does not, however, prove, apart from other indications, that the lesion is organic; or, if organic, that it is situated in the cortical motor zone. I have found experimentally, and it is also in accordance with the facts of human pathology, that irritation primarily situated outside the motor zone may result in an attack of unilateral convulsions. But in these cases there is no doubt that the convulsions are proximately due to irritation secondarily set up in the cortical matter of the Rolandic area; since convulsions of the type known as Jacksonian epilepsy—that is, tonic followed by clonic spasms on the opposite side of the body—never occur on stimulation of the medullary fibres alone, and cannot be produced if the cortical motor centres are destroyed on both sides. Irritation of the subjacent medullary fibres causes tonic contractions only, and the duration of the effect is strictly proportional to that of the stimulus which is applied.

In the great majority of instances the lesions which cause Jacksonian epilepsy are situated either in the cortex itself or immediately subjacent. There is reason to believe, however, that an irritable or explosive condition of the cortex manifesting itself in unilateral spasms, monoplegic or hemiplegic, may be set up by lesions primarily situated in the periphery. Thus Mills (47) has recorded a case of epilepsy of the Jacksonian type in connection with a neuroma of the right hand, the result of injury. The spasms affected the right side, beginning with the fingers, and extending to the arm, face, and leg without loss of consciousness.

Convulsions of a similar peripheral origin have been produced by Brown-Séquard by lesions of the sciatic nerve in guinea-pigs. It is also worthy of note that unilateral convulsions may be caused on the same side by irritation of the dura mater. This I have frequently seen in the course of my experiments.

The *morbid anatomy of Jacksonian epilepsy* is variable, but, from the point of view of regional diagnosis, the exact character of the lesions is unimportant. Generally, it may be said, they are such as are calculated to produce an inflammatory or hyperæmic condition of the cortical centres. They include exostoses, depressed fractures, spicula of bone and cicatrices, resulting from traumatic lesion, meningeal irritation, tuberculous and other tumours, and especially syphilitic lesions of the cortex. These last are such a frequent and potent cause of Jacksonian epilepsy that syphilitic epilepsy and Jacksonian epilepsy are sometimes used interchangeably, though the one name is by no means coextensive with the other.

As has been remarked before, the disease, which begins as an irritative lesion, very generally tends to destroy the region in which it is situated, and thus leads to paralysis with secondary degeneration; irritative phenomena being prone to occur so long as the vitality of the gray matter is not entirely exhausted. It is this tendency which enables us to arrive at the regional diagnosis of the seat of the lesion in a more precise manner than if the phenomena were of the purely irritative order; for the determination of the seat of an irritative lesion is more uncertain than that of a destructive lesion, owing to the difficulty of determining the exact extent of the zone through which a vital irritation may diffuse itself.

If, however, the convulsive discharge be associated with, or followed by, paralytic weakness in the face, arm, or leg, the position of the lesion can be more accurately determined, according to the principles which will be laid down more fully in the next section.

Jacksonian epilepsy in its early stages, and often throughout its whole course, is limited to the face, arm, or leg; or to some particular muscular group on the opposite side of the body. With this monospasm or protospasm there is often a tendency to generalisation, and the convulsions usually follow a definite order. If the spasms begin in the face, they next attack the arm, and then the leg; if they begin in the hand, they ascend the arm, then attack the face, and ultimately spread to the leg; if they begin in the leg, they next affect the arm, and lastly the face. This order is rarely inverted, and it never happens that a spasm, beginning in the face, attacks the leg before affecting the arm. The explanation of the sequence of the spasms in Jacksonian epilepsy is afforded by the position in the Rolandic area of the respective centres of the face, arm, and leg. In order that irritation starting from the facial area should ascend to the leg area, it is necessary that it should diffuse itself through the arm area which separates them from each other. Usually, when the convulsions, starting in a given point, have become unilateral, consciousness is lost; and in this case the convulsions generally extend to the other side, and become bilateral.

Three types of Jacksonian epilepsy may be conceived, according as the spasms begin in the face, arm, or leg.

First, *facial monospasm or protospasm.*—In this case the spasms usually begin in the angle of the mouth, spread thence to the head and neck, causing rotation and conjugate deviation to the opposite side, and thence tend to spread to the arm and become general. Under such conditions we may assume the existence of an irritative lesion of the facial centre situated at the lower extremity of the Rolandic area. It is not necessary here to refer in detail to the evidence in support of this proposition. The chief facts bearing on this subject may be found on reference to Charcot and Pitres (13), Grasset, De Boyer, and the author's *Localisation of Cerebral Disease* (23).

In an interesting case reported by Dr. Byrom Bramwell (10) the fits began with spasm in the right platysma, and were frequently confined to this muscle. The lesion in this case was a small spiculum of bone projecting from the inner table of the skull, the result of a cranial injury received some years previously, which impinged on the cortex at the inferior extremity of the ascending parietal convolution. The situation of the lesion exactly corresponded to the position of the centre for the platysma, as defined by electrical irritation of the brain of the monkey.

Secondly, *brachial monospasm or protospasm.*—Of cases of spasms, dependent on cortical lesion, beginning in the hand and arm, or limited to it, there are now many on record. As a rule they begin in the fingers, and more especially in the thumb and index finger; but this is not necessarily so, as much depends upon the exact position of the lesion in the arm area.

As has already been noted, the proximal movements of the arm are represented in the upper part of the arm area, and the distal in the lower. In some recorded cases no exact description is given of the march of the spasms; but in others, in which they specially began in the fingers or hand, the lesion has been found either in the ascending parietal convolution, or in close proximity to it.

One of the most interesting cases of this kind has been reported by Dr. Dreschfeld. The patient suffered from repeated attacks of convulsions limited to the left arm, beginning with closure of the fist, flexion of the wrist and pronation of the forearm, together with spasms of the left platysma. The dura mater was found adherent to the cortex on the right side over the greater part of the ascending parietal convolution and supramarginal lobule. In a case under the care of Dr. Hughlings Jackson, reported by Mr. Horsley (35), the patient was subject to spasms beginning in the left thumb and forefinger, and frequently followed by temporary paralysis of the left arm. The spasms depended upon the existence of a small tumour situated at the junction of the lower and middle thirds of the posterior central convolution in the right hemisphere. The lesion was accurately diagnosed from the symptoms, exposed, and removed. In those cases in which the spasms have begun in the upper arm and descended to the hand, the lesion has been found at the upper part of the arm area. A typical case of this kind has been recorded by Hughlings Jackson.

In these and similar facts we have reason to believe that the exact situation of the lesion in the arm area may be determined by careful observation of the mode in which the attacks set in; and that, in accordance with electrical stimulation, the spasms will begin in the proximal or distal part of the limb, according as the lesion is situated in the upper or lower division of the arm area.

Thirdly, *crural monospasm or protospasm.*—In the third group of cases the fits begin in the lower extremity, and may begin in the foot and ascend to the thigh, or in the muscles of the thigh and descend to the foot. In such cases the lesion is found at the upper extremity of the Rolandic fissure, as in a case reported by Hughlings Jackson. In another reported by the same author the fits began in the right leg, and were frequently limited to it, followed in time by paralysis of this limb and other signs of extension to the hemisphere in general. A tumour was found at the upper extremity of the ascending frontal and base of the superior and middle frontal convolutions. Many similar instances might be mentioned. For further details the reader is referred to the author's Goulstonian and Croonian lectures (23).

The monospasms or protospasms of Jacksonian epilepsy are not infrequently preceded, or accompanied by, a subjective sensation or sensory aura in the parts invaded by the spasm. It may be limited to these, or the sensory discharge may be more extensive. Thus, in a case reported by Ransom, the fits began with tingling in the left thumb, followed by spasms which spread up the arm to the face. Occasionally the tingling sensation occurred alone, without spasm; and in addition, at a later date, there was also a visual aura in the left eye. The lesion in this case was an adhesion of the pia mater to the cortex at the junction of the middle with the lower third of the Rolandic area of the right hemisphere.

Confirmatory evidence of the regional diagnosis founded on the symptomatology may in many cases be obtained by deep pressure on the skull over the suspected seat of the lesion. Tenderness or distinct pain may thus be elicited, even though the patient may not have complained spontaneously of pain in this region. This is particularly true of syphilitic and irritative lesions in general. In the case of lesions, however, which do not actually reach the cortex, this symptom may be entirely absent.

Summary.—(i.) Irritative lesions of the Rolandic area are indicated by epileptiform convulsions on the opposite side of the body, which may be limited to the arm, leg, or face; or, beginning in this way, may become general, with or without loss of consciousness.

(ii.) There need not be any discoverable organic lesion; but if, in addition to the muscular spasms, there is paralysis more or less enduring, and tending to become permanent, we may diagnose an organic lesion.

(iii.) The nature of the lesion, apart from external indications, must be determined by the characters peculiar to each, such as tumour, syphilis, and the like; and its exact seat by its limitation or mode of onset in the face, arm, or leg respectively.

(iv.) If in the face, the lesion is situated in the lower third of the Rolandic area, or in close proximity to it. If in the arm, in the middle third; if in the leg, at the upper third of the Rolandic area: and it may be more exactly defined in these respective areas according to the muscles primarily thrown into spasm.

(v.) Confirmatory of the evidence derived from the mode of onset of the convulsions is the fact that pain may frequently be elicited by deep pressure over the corresponding cranio-topographical area.

From these indications the seat of the disease may as a rule be accurately defined with a view to excision of an epileptogenic focus.

Destructive lesions.—A. *Affections of motion.*—Destructive lesions of the Rolandic area are the counterpart of irritative lesions, and manifest themselves in paralysis of voluntary motion, limited or general, according as the lesion invades special areas or the whole Rolandic region. This is true of all lesions which positively destroy the cortical gray matter. The apparent exceptions to this rule have been cases of superficial lesion, or tumours growing in or on the cortex, which, it is well known, may in the course of their growth gradually push aside, without actually destroying, the true nerve structures in which they have their seat. Cases of this kind have been reported by Samt, Magnan, Bramwell (11), Bennett, and others. Or the lesions may have caused a certain amount of destruction, but the symptoms during life have either not been carefully observed, or the lesions have occurred before the brain had reached its full development, thus allowing of a greater degree of compensation by the motor centres of the other hemisphere, or by the subjacent ganglia. In this group may be classed the case recorded by Cunningham, and cases of porencephaly, or infantile hemiplegia, in many of which the paralysis is less in degree and extent than is usually found as the result of similar lesions in adults.

Hemiplegia of the cortical type, due to destruction of the whole Rolandic area, does not differ essentially from hemiplegia due to destruction of the anterior part of the posterior limb of the internal capsule (see p. 330). There is the same relative affection of the different movements, those being most paralysed which are most volitional, and least associated with those of the other side of the body. The facial paralysis is most marked in the lower facial region, and less so, but still evident on careful examination, in the upper facial region. The movements of the leg are less paralysed than those of the arm, and the proximal movements of the arm less than those of the wrist and fingers; while those of the trunk, articulation, and phonation are not perceptibly affected.

If the destruction of the cortical substance be complete the paralysis will be of permanent duration, and followed sooner or later by secondary contracture of the paralysed limbs, with descending sclerosis of the pyramidal tracts. If the lesion is not such as to destroy the gray matter completely, the hemiplegia may be transitory, as is not infrequently seen in the course of general paralysis, or of superficial meningo-encephalitis. A complete hemiplegia, however, of greater or less duration does not necessarily indicate total destruction of the whole motor area, for the lesion may have anatomical limits, while the paralysis on the opposite side is general. In such cases we have to deal with merely dynamic disturbances, extending beyond the actually demonstrable lesion; and in the course of time the parts which have not been actually destroyed may regain their functions, and the paralysis become restricted.

Though the rule is that the paralysis occurs on the opposite side of the body, there is no doubt that in some rare instances the paralysis has been direct, that is, on the same side as the lesion. These cases, however, are by no means so numerous as Brown-Séquard (12) has represented, for a large proportion may without doubt be set down to faulty records. But that they may occur has been proved conclusively by a recent case carefully observed and recorded by Charcot and Pitres (14). This was a case of left hemiplegia with aphasia occurring suddenly without loss of consciousness, and without affection of sensation. The paralysis and aphasia existed a whole year. After death the right hemisphere was found intact. In the left hemisphere a large area of softening was found extending from the ascending limb of the Sylvian fissure into the lower part of the ascending frontal, the corresponding portion of the ascending parietal, and the base of the second frontal convolutions. The lesion was limited below by the horizontal limb of the fissure of Sylvius; and sections through the softened region showed that the lesion involved only the cortex and subjacent medullary fibres, and did not invade the internal capsule or basal ganglia. Secondary degeneration was traced into the left crus and the left pyramid. In this case there was no decussation of the anterior pyramidal tracts in the usual situation. Under such conditions a direct paralysis is easily understood and accounted for.

Complete hemiplegia, due to destructive lesion of the Rolandic area, does not differ essentially from that caused by lesion of the corresponding part of the internal capsule. This statement, however, requires qualification in respect to the left hemisphere. In this case, in the vast majority of instances, the paralysis of the limbs and face is associated with aphasia—an association which at once distinguishes cortical from capsular right hemiplegia.

Hemiplegia of the ordinary kind, however, is not the most common form of cortical paralysis, for only rarely can the whole motor cortex be simultaneously destroyed by any pathological or traumatic lesion. Necrotic softening, from vascular occlusion of the middle cerebral artery, still leaves intact the upper part of the Rolandic area supplied by the anterior cerebral; and a simultaneous blocking of these two channels, though conceivable, is practically an unknown occurrence. Hence in the completest form of cortical hemiplegia from vascular occlusion the lower extremity is comparatively little affected. More often complete hemiplegia of cortical origin is a succession of monoplegias, due to extension of a lesion beginning in some definite area; and we may diagnose the primary seat of the lesion by a consideration of the movements which were first paralysed and the mode in which others were successively abolished.

Crural monoplegia.—If the paralysis is limited to the leg, or begins in it, the primary seat of the lesion is the paracentral lobule or upper extremity of the central convolutions adjoining the longitudinal fissure. In this form of monoplegia—crural monoplegia—of which there are now many cases on record, traumatic and otherwise, the lesion has been found in the region indicated. This region appears to be a favourite seat of circumscribed tuberculous disease. (On this, *vide* case reported by Souques and Charcot (64); a similar one by Ballet; and another by myself (207).

The frequency of tuberculous disease in this region is attributed by Souques and Charcot to the relative richness of its circulation, as it derives blood from both the Sylvian and anterior cerebral arteries. This also is the region more particularly involved in birth palsy, or congenital spastic paraplegia due to traumatic meningeal hæmorrhage (M'Nutt). Crural paralysis is very frequently associated with a greater or less degree of paralysis of the arm also, constituting the mixed type, *brachio-crural monoplegia.* In at least ten cases of crural monoplegia, which I have found on record, the lesion occupied the paracentral lobule, and the adjoining convex aspect of the hemisphere at the upper third of the Rolandic zone. And in many of the combined cases the progress of the disease from above downwards was distinctly traced by the course of a paralysis beginning in the leg and gradually extending to the arm.

Brachial monoplegia.—Paralysis limited to the upper extremity or to some of the movements of this limb, constituting brachial monoplegia, is of very common occurrence. The lesions, with some variation in their extent, have been found to implicate the middle two-fourths of the Rolandic area, and the evidence is in favour of the lower and posterior portion being especially related to the finer movements of the hand and wrist. It is necessary, however, in this connection to remember that a lesion primarily situated outside the motor area and gradually invading it, such as a tumour, is apt first of all to cause weakness of the wrist and fingers—an illustration of the general principle that the finer or more specialised movements are the first to give way before an invading or destroying lesion.

Many of the cases of brachial monoplegia on record have been the result of excision of a supposed focus of Jacksonian epilepsy; some of these cases will be referred to subsequently in connection with another problem (p. 299).

Brachial monoplegia is frequently associated with paralysis of the face of the ordinary cerebral type, constituting the combined form, *brachio-facial monoplegia;* an association specially apt to occur from occlusion of the Sylvian artery, and as a rule associated with aphasia when the lesion is in the left hemisphere. This associated paralysis is owing to the implication of the facial with the brachial centres; and in at least twenty cases of this kind, which I have investigated, the lesion was found to invade the lower half of the Rolandic zone. These cases are so common that it is unnecessary to refer to them in detail. If paralysis of this form on the right side is not associated with aphasia the probability is that the patient is left-handed; or that the lesion is not cortical, as in a case reported by Dieulafoy.

Facial monoplegia.—Facial paralysis of the cerebral type—facial monoplegia,—that is, paralysis limited to the lower facial region, uncomplicated with paralysis of the arm, is not a very common occurrence. Usually it is associated with some affection of the tongue and muscles of articulation. One of the earliest cases of cortical facial paralysis on record has been reported by Hitzig. This was the case of a soldier who, after a bullet wound on the right side of the head, began to have spasm in the left side of the face and tongue, and to a slight extent also of the left hand. After death an abscess was found at the lower extremity of the right ascending frontal convolution, between the precentral sulcus and the fissure of Rolando. In an interesting case of left facial spasm of the Jacksonian type, under my own care, operated upon by Mr. Horsley, excision of the lower extremity of the right ascending frontal convolution caused weakness of the left side of the face and defective articulation.

The lowermost extremity of the ascending frontal convolution, and base of the third frontal, is the region in which stimulation causes movements of the tongue, of the muscles of articulation, and also of the larynx. Physiological experiments show that these centres have bilateral relations, stimulation causing movements of both sides. Hence it is that unilateral lesion causes only slight or almost imperceptible weakness on the one side. This fact explains also why the lesion which causes aphasia does not imply paralysis of articulation or phonation.

It is not necessary, in this section, to discuss the pathology of aphasia further than to state that the aphasia here referred to is Broca's or motor aphasia; that is, the inability to express ideas in articulate speech, while ideation in general, and the comprehension of speech spoken and written, remain unimpaired. Usually the lesion causing aphasia is vascular obstruction of the left middle cerebral artery; and it has been supposed by some that the greater frequency of aphasia with right than with left hemiplegia is due to the greater frequency of vascular occlusion in the left hemisphere, as compared with the right. I have elsewhere (23) shown that this surmise is completely disposed of by the reported cases of aphasia from traumatic lesions at the lower extremity of the Rolandic zone. For references to such instances see Sidney Jones, Simon, Proust and Terillon, MacCormac, Macewen.

As already stated, in cases of motor aphasia there is no paralysis of the muscles of articulation, but at most a slight weakness of the right angle of the mouth and slight deviation of the tongue to the right. If, however, the lesion is bilateral, not merely is there aphasia, but also paralysis of the muscles of the tongue and articulation. The best instance of this kind on record is a case reported by Barlow. A boy aged 10, the subject of aortic disease, of which he ultimately died, was seized with right hemiplegia (chiefly brachio-facial) and aphasia. From this he recovered at the end of a month. Three months later he was seized with left brachio-facial monoplegia. With this attack there occurred aphasia also, together with paralysis of the movements of the mouth and tongue. He was unable to show his teeth, or to protrude his tongue; and there appeared, in the words of Dr. Barlow, "to be loss of voluntary motor power over the muscles concerned in deglutition and articulation; reflex deglutition, however, was unimpaired. Mastication was difficult, but not entirely paralysed, and the patient was able to phonate. There was no affection of sensation in the paralysed parts, either in the skin or mucous membranes, and the muscles reacted normally to the faradic current." The condition lasted till death. After death a patch of softening was found in each hemisphere, occupying the lower end of the ascending frontal, and the hinder end of the middle and inferior frontal convolutions (3). The condition existing in this case is in many respects like that met with in bulbar paralysis, though there are some essential points of difference which will be discussed again, in connection with a similar group of symptoms termed "pseudo-bulbar" paralysis, to distinguish it from that due to nuclear lesion of the medulla oblongata.

As has been mentioned, stimulation of the lower extremity of the ascending frontal convolution causes phonatory movements of the vocal cords. The effect here is distinctly bilateral; it is not to be expected, therefore, that unilateral lesion should cause any appreciable affection of the movements of the vocal cords. It seems somewhat strange, however, that in Barlow's case phonation was still possible, notwithstanding the apparent total destruction of the region in which the laryngeal movements are centralised. If the centres in question were entirely destroyed, we must assume that, as in the case of the oculo-motor centres, compensation may be effected by the basal ganglia. Some cases have, however, been reported in which unilateral cortical lesion in the region indicated was associated with paralysis of the opposite vocal cord. In one of these, by Seguin, there was, apparently in connection with lesion of the posterior part of the third frontal convolution on the right side, some affection of phonation; and in another, by Garel and Dor, there was paralysis of the left vocal cord, apparently in connection with a lesion of the right hemisphere at the base of the third frontal convolution. The cases, however, were not verified by laryngoscopic examination, nor was the possibility of a bulbar or peripheral nerve lesion disproved; and it is exceedingly improbable, in view of the facts of Barlow's case, together with those of experimental physiology, that paralysis of the vocal cord can be produced by a unilateral cortical lesion. Moreover, Sir F. Semon has shown, by laryngoscopic examination of cases of right hemiplegia and aphasia within a few hours of the attack, that both vocal cords move in a perfectly normal manner.

While, therefore, it is probable that the laryngeal centre is situated at the lower extremity of the ascending frontal convolution, there is no satisfactory evidence on record that complete paralysis of one or both cords has been caused by cortical lesion.

The same may be said of the muscles of mastication. Movements of the same muscles are also concerned in the act of articulation, and these are excited bilaterally by stimulation of the cortex. Paralysis of these muscles cannot, therefore, be expected from anything less than a bilateral lesion in corresponding parts. In Barlow's case, however, though the movements of mastication were excessively feeble, they were not entirely paralysed.

Lepine, Langer, and Picot (quoted by Charcot and Pitres) have observed trismus in connection with cortical lesions; but the pathology of this symptom, with its bearing on the position of the masticatory centre, has not been established. A case has been reported, by Hirt, in which masticatory paralysis was apparently due to cortical lesion; but the case was a complicated one, for, in addition to multiple cortical lesions, there was degeneration in the spinal cord also.

We may feel assured that every movement under the control of the will is represented unilaterally or bilaterally in the Rolandic area; but the clinical evidence at present available does not enable us to indicate the centres of any others beyond those already discussed in the preceding pages.

B. *Affections of sensation.*—Cortical paralysis due to destructive lesion of the Rolandic area is essentially a motor paralysis, and need not be accompanied by loss of sensation, cutaneous or so-called muscular.

The patient whose limbs are entirely paralysed from cortical disease may feel and localise the slightest touch, appreciate temperature, and correctly indicate every movement communicated to his paralysed limbs. Thus, in a case of aphasia and right hemiplegia under my care in the National Hospital for the Paralysed and Epileptic, the patient, whose right arm, a month after the onset, was still absolutely paralysed, felt and localised the slightest touch on the paralysed limb, and correctly indicated with the left hand every position passively communicated to the right. After death necrotic softening, due to blocking of the Sylvian artery, was found in the Rolandic area of the left hemisphere. But though, as the above case proves, paralysis of cortical origin may occur without the slightest affection of sensation, it is undoubtedly true that affections of sensation of a greater or less extent are met with in connection with lesions apparently confined to the cortex of the Rolandic zone. And it is a question on which there is still considerable difference of opinion, whether sensation is not also represented, along with motion, in what is usually designated the motor area of the cortex.

An analysis of 284 recorded cases of lesion of the Rolandic zone, which I made in 1890 (*Croonian Lectures on Cerebral Localisation*), showed that in 100 the state of the sensibility was not mentioned; in 121 it was noted to be intact, and in many of these every variety of sensibility was expressly stated to have been carefully investigated. In 63 some impairment of sensibility was noted; in 28 of these, however, the lesion was not strictly confined to the Rolandic area, but implicated the adjacent lobes, especially the parietal. Among the remaining 35 conditions existed, in the majority at any rate, which were calculated to implicate centres or tracts demonstrably connected with the conveyance or perception of sensory impressions. Similar conclusions were arrived at by Charcot in the work on which he was engaged with M. Pitres just before his death, and are summarised in the *Archives cliniques de Bordeaux*. Sept. 1894.

"In our opinion the anæsthesiæ which sometimes accompany motor paralysis of cortical origin are most frequently functional, and are analogous to, if not identical with, hysterical anæsthesia. The discussion of the reasons which appear to us to justify this opinion would take us far beyond the limits which we have set ourselves in this chapter. We content ourselves, therefore, with stating that, from an examination of the facts of clinical pathology, the anæsthesiæ which sometimes accompany paralysis of cortical origin are unessential and accidental phenomena, and do not depend directly on lesions of the Rolandic zone, and play no pathogenic part in the development of paralytic symptoms" (p. 43).

Nothnagel, writing in 1879 (51), arrived at the conclusion that "up to the present there is no case in which, with a purely superficial or purely cortical lesion, persistent anæsthesia has been observed. In all cases the medullary substance has also been affected, and it is not improbable that in such cases the sensory tracts of the internal capsule may have been injured directly or indirectly. For the diagnosis, therefore, of superficial lesions, disturbances of sensation have no importance" (p. 473).

It is maintained, on the other hand, by many writers—Petrina, Exner, Luciani and Sepilli, Starr (65), Dana (16), and others—that the facts of disease prove that the sensory centres coincide with the motor centres in the Rolandic zone; by Horsley, Beevor, and others, that sensation is to some extent represented in the Rolandic zone; while Bastian regards the Rolandic zone as the seat of kinæsthesis or so-called muscular sense.

As the coincidence of the sensory with the motor centres in the Rolandic zone is an opinion which is largely entertained by clinical observers, it appears necessary to consider somewhat in detail the facts on which it is based. As has already been stated, there is no doubt that in many cases of lesion of the Rolandic zone, anæsthesia has been observed coinciding to a greater or less extent with the motor paralysis. The question, however, is whether the affection of sensation is a direct result of the cortical lesion, or only an indirect or dynamic influence on sensory structures anatomically distinct from the motor centres proper. In this relation tumours, and all such lesions as are known to cause indefinite indirect effects, are of no value as evidence, and may be summarily excluded. Since attention has been specially directed to this point, many cases of excision of portions of the motor cortex for the cure of focal or Jacksonian epilepsy have been put on record, and it is of importance to consider their bearing on the question.

In a case by von Bergmann, of excision of a considerable portion of the hand centre in the left hemisphere, there was not the slightest affection of sensibility in any of its forms, muscular or otherwise. In a case by Keen (40), after excision of the centre for the movements of the hand and wrist in the right hemisphere (as determined by faradic stimulation), the left hand was paralysed in all movements both of the fingers and wrist, and the forearm was paretic; while the shoulder and face were unaffected. On the following day in the left hand each finger was recognised correctly, but the two points of the æsthesiometer were recognised as only one even when separated as much as the length of the finger or the entire breadth of the hand. The right hand, however, was but little better, as the two points were recognised as one at two-thirds of the length of the fingers apart, and about the same crosswise.

In a second case, by the same author, of excision of a portion of the right motor cortex injured by a depressed fracture, the left fingers and wrist were paralysed. On the second day following the operation on the right fore and little finger, the two points of the æsthesiometer were appreciated as one at $\frac{1}{16}$ of an inch, while on the corresponding fingers on the left side only at $\frac{6}{16}$ of an inch. On the twelfth day there was still slight impairment of sensation in the middle of the forearm and third and fourth finger, but the position of the æsthesiometer was clearly indicated. This condition as to sensibility was similar to that which existed before the operation.

Lloyd and Deaver (41) report a case of excision of a portion of the hand centre of the right hemisphere, which caused paralysis of the flexors of the left wrist and fingers, and of the left half of the face. Three months after the operation, while there was still considerable paresis of the left hand, the patient was able to feel the slightest touch with the blunt points of an æsthesiometer, though localisation was somewhat imperfect. With weights varying from two to twelve ounces, he was able to tell the heaviest, by cutaneous pressure, as well on the affected as on the sound side. He could not distinguish the form of objects on account of his inability to move his fingers. Sensation to pain and heat was perfect.

In a case under my own care (27) excision of a traumatic cicatrix at the upper extremity of the left Rolandic area was followed by slight loss of tactile sensibility on the dorsum of the two distal phalanges of the right hand, and slight inability to indicate correctly the position passively communicated to them. Elsewhere, and on the right foot which was equally paralysed, sensibility was normal. Ultimately the impairment of sensibility disappeared entirely, while the fingers remained feeble and rigid. In this case the lesion was such as was calculated actually to implicate a portion of the gyrus fornicatus (27). In the case of T. W. (27*a*), after excision of a small tumour, which spread widely under the cortex, at the lower part of the ascending parietal convolution of the right hemisphere, there was observed on the fourth day total paralysis of the muscles of the left hand, together with tactile anæsthesia over the whole of the left side of the body, and inability to tell the position in which his thumb, or fingers, or elbow were placed. Seven weeks afterwards he had entirely recovered the power of movement except in the hand; and he was not always correct as to the position of the thumb or fingers on passive movements with the eyes shut. Ten days later, evidently from extension of the primary disease, the left limbs became paretic, tactile and painful; sensibility became lost in the left leg as high as the knee, and tactile sensation absent in the thigh. Tactile and painful impressions were appreciated over the whole of the trunk, but tactile sensibility was absent in the left arm, and the sense of pain defective. Tactile sensibility was also absent on the left side of the face, together with some degree of analgesia. A case has been reported by Mills and Keen (48) of excision of the whole of the centre for the movements of the upper arm in the right hemisphere, the position of which was correctly determined by faradic stimulation. After the operation there was entire paralysis of the shoulder movements of the left arm, and weakness of the other movements of the limb. This paralysis increased so that on the tenth day there was total paralysis of the left arm and almost complete paralysis of the left leg. Slight improvement began to appear after this date, so that on the thirty-third day only some degree of weakness remained. In this case all forms of sensibility were accurately tested by Mills, with the result of showing that, as regards the power of perceiving and localising tactile impressions, there was never the slightest defect. The muscles on the paralysed side were active to faradic stimulation, and the patient could recognise accurately the different positions in which her limbs were placed.

In a case of focal epilepsy reported by Dr. Hale White, a portion of the cortex corresponding to the arm centre in the left ascending parietal convolution was deeply cut away, so as to expose the medullary fibres. This was followed by paralysis of the face, arm, and leg on the right side, together with aphasia. There was not the slight defect in the perception or localisation of tactile impressions.

A similar case is recorded, by Rushton Parker and Gotch, of excision of a portion of the genu of the ascending frontal convolution of the right hemisphere. This was followed by temporary loss of power in the left hand, but without any impairment of sensibility.

A case of excision of a portion of the cortex has also been reported by Bidwell and Sherrington. The portion removed was from the region stimulation of which caused movements of the foot and knee. This was followed neither by obvious impairment of motion nor of sensation.

In a case under my own care (27), of excision of a tumour from the Rolandic zone of the right hemisphere, the left arm and leg were completely paralysed and the left side of the face partially so. Sensation was practically normal over the whole of the left side, though sometimes the patient referred a touch to the nearest joint above the part stimulated. This, however, was not always the case, as he sometimes indicated the part touched quite correctly. Muscular sense was good, and he was able to indicate the position of his limbs when passively moved. Beevor and Ballance report a case of removal of a tumour involving the upper part of the ascending parietal convolution, the anterior part of the parietal lobule, and the adjacent marginal gyrus. Seven days later there was complete paralysis of the right arm, and a paretic condition of the right leg. The slightest touch was perceived on the right side, but not accurately localised. Within five months after the operation there was still paralysis of the toes and loss of dorsi-flexion of the ankle, but sensation was everywhere perfect.

Diller and Buchanan have reported a case of paresis of the right limbs and right side of the face, due to a subcortical cyst in the lower part of the left ascending frontal convolution. The patient could feel and accurately localise a touch on any part of the paretic side, and could discriminate weights accurately. After removal of the cyst there was some improvement in the paretic condition, and there was no affection of sensation. Excision of the facial centre in the right hemisphere in a case under my own care caused paralysis of the left side of the face without any impairment of sensation.

Albertini and Brigatti report a case of removal of a glioma the size of a hen's egg from the middle of the right Rolandic zone, which caused paresis of the left side, and epileptiform spasms of the left arm and left angle of the mouth. Over a year after the operation there was paresis of the left side of the face, paresis of the arm, and complete paralysis of the hand and foot. The sensibility of the left side, which before the operation was normal, was diminished for all qualities of sensation. The muscular sense in the left arm was lost.

Starr and M'Burney record a case of right hemiplegia and aphasia from a traumatic lesion of the left hemisphere. The right arm and leg were almost totally paralysed. The muscular sense test, it was said, could not be satisfactorily applied on account of the patient's apathetic condition. On trephining, a clot of blood was removed which extended from the posterior third of the left frontal convolution over the ascending frontal into the fissure of Rolando. Recovery took place, but the right hand remained paralysed, the fingers being flexed and rigid. In the hand there was marked diminution of tactile and painful sensibility, two points being felt as one at two centimetres apart on the tips of the finger. The muscular and temperature senses were perfect.

Other similar cases might be quoted, but it seems unnecessary to multiply instances as the lessons they teach are essentially the same. Analysis shows that there are some variations in the condition of sensation after cortical lesions of the Rolandic area. In the majority of pure cortical lesions there was not the slightest affection of sensibility in any of its forms from the first, though there was marked motor paralysis; while in some the slight affection of sensation which was at first observable ultimately disappeared. This was true of Beevor and Ballance's case, where, notwithstanding the permanent paralysis of the hand and foot, sensibility completely returned. In Starr's case the persistence of defective tactile and painful sensibility at the tips of the fingers may be ascribed to the rigid flexion of the fingers; and in others, such as

Albertini and Brigatti's—as the lesions were of the nature of tumours extending deeply into the brain substance—there was at least the possibility of implication of the sensory tracts.

It is evident from the facts above narrated that, at any rate, relatively small portions of the convexity of the Rolandic zone may be removed without causing the slightest impairment of tactile or cutaneous sensibility in any of its forms, though the lesion be sufficient to produce more or less extensive paralysis. It is probable, therefore, that such affections of sensation as may occur from destruction of a larger area are of a merely dynamical character, if the lesion do not implicate the sensory tracts or centres. If there were any necessary relation between the sensory defect and the motor paralysis the two should coexist and be equally enduring, but this is not the case; for the defect of sensibility, at first observable, gradually lessens and ultimately disappears.

The order in which sensibility normally returns in cerebral hemianæsthesia explains the occasional apparent limitation of the sensory defect to the limb most paralysed. The affection of tactile and general sensibility follows the same rule as the distribution of muscular paralysis; that is, it is most marked, and endures longest in the parts most volitional. Hence when cerebral hemianæsthesia is beginning it shows itself first in the fingers, and disappears last from this part. A certain degree of tactile defect may therefore be observed in the hand, when the face, trunk, and leg have entirely recovered. The remnant, therefore, of a general hemianæsthesia, when associated with partial hemiplegia or brachial monoplegia, may bring about the coexistence of brachial monoplegia with brachial mono-anæsthesia, apparently from the one cortical lesion; but in reality this effect is of complex causation.

Most of those who hold that the motor and sensory centres coincide in the Rolandic area are obliged to admit that they are not coextensive, and to assume that the sensory centre of any given part occupies relatively a much larger area than its motor centre. Hence, for instance, in order to cause anæsthesia of the face, the lesion must be such as to cause also motor paralysis of the arm or leg or more; that is, that the sensory centre of the face embraces also the motor centres of the arm and leg. Such a hypothesis on the part of those who otherwise advocate a localisation of function in the cerebral cortex appears to me rather absurd.

That clinical and experimental lesions of the Rolandic area of considerable extent should temporarily cause some impairment of sensation on the paralysed side may be admitted without the necessity of assuming that the sensory centres are localised in the same region.

The experiments of Schiff, Munk, Tripier, Luciani and Seppili, Mott, and others may be accepted as proving that some impairment of sensation may be caused by lesions apparently restricted to the motor zone; but that these are only dynamical disturbances is, I think, shown by the results of certain experiments on monkeys which I have recently performed with my colleague Dr. Turner.

In several instances we destroyed the Rolandic area, wholly or partially, in such a way as to remove the cortex in the most thorough manner, both on the convexity of the convolutions and in the depths of the sulci. This was followed in one case by total hemiplegia of the opposite side, great impairment of tactile sensibility, and hemianopsy. The destruction of the Rolandic area did not extend into the facial centre, but the anæsthesia affected the face equally with the parts that were paralysed. In the course of a few days, however, while the motor paralysis continued, the anæsthesia and hemianopsy gradually diminished, the last remnant of anæsthesia being the inability to localise correctly a small ivory clip exerting slight pressure on the skin of the fingers or toes.

In another experiment, in which the upper third of the Rolandic area was entirely destroyed in the left hemisphere, the lesion extending down to the calloso-marginal fissure on the mesial aspect, there was almost total paralysis of the right leg, and to a considerable extent also of the right arm. Together with this motor paralysis there was loss of tactile sensibility, without analgesia, over the whole of the right side—ear, face, arm, and leg. Within three days, however, this had almost entirely disappeared, so that the animal, blindfolded, was evidently aware of drops of water allowed to fall on its toes, as it put its hand at once on the spot.

It is obvious from such facts that the sensory and motor centres are not coincident in the Rolandic area, inasmuch as practically the whole of this area may be removed without permanent loss of sensation. It may, however, be said that the recovery in such case is due to bilateral association and the compensatory action of the centres of the sound side. This is the hypothesis which is advocated by Brissaud. If this hypothesis were correct there should have been in Barlow's case (p. 296) bilateral anæsthesia of the face, inasmuch as both facial centres were destroyed. It was expressly stated, however, that sensibility was absolutely normal.

In a case of double brachial monoplegia, recorded by Bourdon, the result of traumatic hæmorrhagic extravasation and softening in the Rolandic area of both hemispheres, it was also expressly stated that sensation was unimpaired throughout.

However, it has been stated, by Dr. Mott (49), that after bilateral extirpation of the leg areas in a monkey the sensibility of the feet was blunt twelve months after the operation. But if the sensory and motor centres of the legs coincide in the Rolandic area, sensibility should have been entirely extinguished in the legs, not merely blunted in the feet. Turner and I found, on the contrary, that after bilateral extirpation of the leg area the animal gradually recovered sensation in the feet, which remained paralysed; so that within a month it could feel and localise the slightest tactile impression on either foot.

The same arguments which are used to establish the localisation of the sensory centres in the Rolandic area might be employed in favour of the localisation of the visual centre in the same region, inasmuch as in connection with a lesion anatomically restricted to the Rolandic area hemianopsy may occur along with hemianæsthesia. And in a case of removal of the whole frontal lobe previously referred to (p. 276), this combination of symptoms resulted. We know, however, that total and permanent hemianopsy can be caused by lesion restricted to the occipito-angular region, and, therefore, any representation of vision in the Rolandic area or frontal lobe may be absolutely excluded; for when the occipito-angular region is destroyed no trace of the visual faculty remains.

Similarly—and inasmuch as it can be proved that sensation may be abolished by cortical lesions altogether outside the Rolandic area—we may argue that there is no representation of sensation in the motor cortex; and that such affections of sensation as may occur in connection with lesions of this region are either merely dynamical or are due to direct implication of the sensory tracts or centres proper.

In several instances, especially those of excision of a portion of the Rolandic area, it has been stated that in addition to some impairment of tactile sensibility there has been loss or impairment of the sense of position or movement; and facts like these have been relied on to prove that the Rolandic area is the seat of the muscular sense. On the other hand, cases have been adduced, by Nothnagel, Luciani and Seppilli, Seligmüller and others, of loss of the muscular sense without motor paralysis, more especially in connection with lesions of the parietal lobe. Redlich, however, shows that the great majority of these have been of such a nature—namely, abscesses or tumours, and so forth—as to exercise an indirect effect upon other parts; and the cases which he himself mentions as having more the character of stationary lesions have all, as he states, been of an extensive character, involving sometimes the upper, sometimes the lower parietal lobe, as well as the angular gyrus and the temporal convolutions or the occipital lobe; and in one case, at least, the loss of the muscular sense was complicated also by hemianopsy. In two of the cases the Rolandic zone was not affected. The same arguments, therefore, which are relied upon to prove that the muscular sense is centralised in the Rolandic area are equally applicable to its localisation in the parietal lobe. It is, however, impossible that both of these contentions can be correct; the probability is that the loss of muscular sense in both instances is merely a coincident disturbance of the functions of sensory tracts and centres situated elsewhere than in the parts actually diseased.

I have never myself met with a case of loss of the sense of position or movement from cerebral lesion in which it was not possible to demonstrate also some impairment of tactile sensibility; more particularly in its highest form, the sense of localisation.

Redlich states that in 132 cases of hemiplegia, examined by him in the Vienna Hospital, he found 28 associated with more or less enduring impairment of the muscular and stereo-gnostic sense—that is, the faculty of distinguishing form; and he considers that this, together with hemianopsy, as has been shown by Sir William Gowers, occurs more often in recent cases of cerebral hæmorrhage, and in all probability from indirect disturbance of the sensory tracts. In all his cases there was some impairment of tactile sensibility also. The impairment of the muscular sense did not, however, run quite parallel to the impairment of tactile sensibility. As a rule the muscular sense was more impaired than the mere perception of tactile impressions; but as regards both the defect was more marked in the limb most paralysed, and more especially in the distal portions of this limb. His observations are in harmony with what has previously been stated, namely, that impairment of tactile sensibility disappears last from the parts which are longest to recover from paralysis after an ordinary attack of hemiplegia. In the great majority of instances in which there was impairment of muscular sense in the hand, tactile sensibility was also impaired over the whole of the corresponding side, as may be assumed from the fact that it was generally impaired in the face. This also is in harmony with the fact, which I have observed, that the impairment of tactile sensibility is often well marked in parts which are not paralysed as to motion.

A case of apparent loss of the muscular sense, apart from tactile anæsthesia, in connection with lesion of the parietal lobe has been recently described by Starr and M'Cosh (67). After trephining for a cranio-cerebral injury of the upper part of the left ascending parietal convolution, a varicose mass of veins on the cortex was tied, and the subjacent tissue probed with a hypodermic needle. The result of this operation was an ataxic condition of the right arm, and inability to indicate the position passively communicated to it. The actual seat of the damage was uncertain, and the case is regarded by Goldscheider (30) as of no value in favour of localising the centre for the muscular sense in the parietal lobe. It can also be shown that lesions have repeatedly occurred in this region without any similar symptom having manifested itself.

Some of the cases of loss of tactile and muscular sensibility, apparently in connection with cortical lesions of the Rolandic area, have been without doubt cases of hysterical hemianæsthesia. What appears to me an exemplary case of this kind, in favour of the localisation of the sensory centres in the parietal and motor cortex, has been adduced by Gowers (31). It is a case reported by Demange, in which there was extensive cortical softening over the whole of one hemisphere. Associated with this was general and special anæsthesia on the opposite side of the body of the usual hysterical type. A similar instance is mentioned by Nothnagel. This was the case of a young man aged twenty-eight, who became suddenly aphasic. At the same time he became completely anæsthetic on the right side, and to a large extent on the left. This condition, however, lasted for a few days only, while the aphasia continued. A lesion was found in the left hemisphere invading Broca's convolution and its neighbourhood (51).

Summary.—(i.) Destructive lesions of the Rolandic area cause paralysis of voluntary motion on the opposite side, general or limited, according to the extent and locality of the lesion. The electrical reactions of the paralysed muscles are not affected. (ii.) When the whole motor cortex is involved the hemiplegia in all respects resembles that caused by destruction of the motor division of the internal capsule. (iii.) Frequently paralysis of cortical origin is dissociated, or consists in a succession of dissociated paralyses or monoplegias. Thus a monoplegia may become a hemiplegia by advance of the lesion from its primary seat. (iv.) Paralysis of voluntary motion of the leg alone, or of the arm and leg, or of certain movements of the arm, or of the arm and face, or of the face alone, may be looked on as dependent upon lesions of the cortex of the Rolandic zone or of the subjacent medullary fibres. (v.) Hemiplegia from cortical lesion of the left hemisphere is almost invariably associated with aphasia. (vi.) Crural monoplegia, or this combined with a greater or less degree of paralysis also of the arm, indicates lesion at the upper extremity of the central convolutions. (vii.) Brachial monoplegia indicates lesion of the middle two-fourths of the Rolandic area. (viii.) Brachio-facial monoplegia indicates lesion of the lower third of the Rolandic area. (ix.) Facial monoplegia (or this combined with aphasia, if the lesion is in the left hemisphere) indicates lesion of the lower extremity of the Rolandic zone posterior to the third frontal. (x.) While it is probable that the centres of movement of the vocal cords, and of the muscles of mastication, are also situated at the lower extremity of the Rolandic area, there is as yet no case on record in which these muscles have been paralysed by cortical lesions, unilateral or bilateral. (xi.) Paralysis of cortical origin may be, and frequently is, entirely independent of impairment of cutaneous or muscular sensibility; and this is the true uncomplicated type of this form of paralysis. Not infrequently, however, and especially in connection with lesions of an extensive and sudden character, or of such a nature as to produce dynamical disturbance of the sensory tracts and centres, it is associated for a time with impairment of sensibility in the paralysed limbs. This rarely extends to the sense of pain or temperature, and is mostly confined to tactile sensibility, and the sense of position and movement. These disturbances are, however, essentially fugacious; and, although at first general, they tend to become restricted to the distal extremities of the paralysed limbs, and ultimately to disappear, even though the motor paralysis remains. (xii.) When the motor cortex is destroyed, secondary sclerosis ensues in the pyramidal tracts, and contracture in the paralysed limbs.

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Lesions of the falciform lobe.—The facts related in connection with lesions of the Rolandic zone, as well as of the parietal lobe, show that neither of these regions can be regarded as the true centre of tactile and muscular sensibility. It is certain that the paths of motor impulse and sensory transmission are distinct from each other in the peripheral nerves, spinal cord, crus cerebri, and internal capsule.

The question is whether the tracts, which have remained separate so far, become fused together in the cortex; or whether they are distributed in definite regions, like those of the special senses?

There is, as yet, comparatively little clinical evidence in favour of the existence of a definite cortical region, lesion of which causes loss of tactile and general sensibility, apart from motor paralysis or other forms of sensory impairment.

Many years ago I showed that destructive lesions of the hippocampal region, or inferior division of the falciform lobe, caused impairment or abolition of tactile and general sensibility, together with a condition of the limbs which indicated loss of the sense of movement without motor paralysis. These experiments were supplemented by those of Horsley and Schäfer, on the gyrus fornicatus (10), who concluded from their experiments that "any extensive lesion of the gyrus fornicatus is followed by hemianæsthesia, more or less marked and persistent. In some cases the anæsthetic condition has involved almost the whole of the opposite side of the body; in others it has been localised to either the upper or lower limb, and to particular parts of the trunk; but we have not yet succeeded in establishing a relationship between special regions of the body and the parts of the convolution which have been destroyed. Moreover, the anæsthesia was frequently very pronounced and general during the first three or four days after the operation, and, indeed, in several instances took the form of complete insensibility to both tactile and painful impressions, so that even a sharp prick or the contact of a hot iron would produce no indication of sensation, but after that time this general condition would become gradually in great part recovered from or more localised in definite regions. In all cases, however, in which the diminution of sensibility was well marked during the first few days, it persisted, although with lessened intensity, for many weeks in those instances in which the animals had been preserved for so long. In other cases in which, apparently, the lesion was slight, the diminution of sensibility, though at first well marked, subsequently disappeared entirely. However, persistent loss of all forms of cutaneous sensibility has never been caused by destructive lesions of the falciform lobe; no doubt because it has been impossible to destroy both limbs of the falciform lobe completely in any one animal."

My recent investigations on this subject, in conjunction with Dr. Turner, have been entirely in harmony with those above mentioned.

The anæsthesia caused by lesions of the falciform lobe is of a much more profound character than that which may be temporarily produced by extensive lesions of the motor area; and this without implication of the motor centres or evidence of motor paralysis. An appearance of motor paralysis is induced, evidently due to the loss of the sense of position and of movement; and the animal tends to slip or fall over on its anæsthetic side: but in climbing the lattice-work of the cage it freely uses the anæsthetic limbs, though the grip both of the hand and foot fails when attention is withdrawn from them.

We have also found, as did Horsley and Schäfer, that the anæsthesia was occasionally much more marked in one limb than in the other; and, in particular, we have found that lesion of the gyrus fornicatus, at the point where it passes into the precuneus, caused loss of sensation specially in the opposite leg, which endured long after it had entirely disappeared from the face and upper limb.

These experiments show that temporary total abolition and long-continued profound impairment of tactile and muscular sensibility may be experimentally induced by cortical lesions of the falciform lobe without motor paralysis, and without impairment of any of the forms of special sensibility. I have myself no doubt that it is to implication of this centre, or of its connections with the posterior tracts of the internal capsule, that the sensory impairment is due which has been observed in association with lesions apparently restricted to the motor zone or parietal lobe.

There are, however, very few cases on record of lesions restricted to the falciform lobe, as this part of the brain seems less liable to injury or disease than the convexity.

Nor should we, from the facts of experiment, be led to expect any noteworthy defect of cutaneous sensibility except in connection with lesions of a considerable extent in this lobe. Among the cases that have been reported of lesion of the gyrus fornicatus the following are worthy of mention :—

Dr. Savill has recorded a case of almost complete loss of sensation on the left side, and partial loss on the right, without any apparent loss of power in the arm or leg. The patient recovered from this attack, and again two years later was readmitted with paralysis of the arm and leg. After death an old hæmorrhagic cyst was found lying underneath the gyrus fornicatus and part of the marginal convolution and anterior half of the quadrate lobule, which passed as far inwards as the roof of the right lateral ventricle. The brain was otherwise healthy.

Dr. Churton has reported a case of anæsthesia of the left foot, without motor paralysis, and without any local vascular or other lesion in this limb, in which, after death, there was found a clot the size of a small walnut in the right calloso-marginal fissure, with erosion and discoloration of the subjacent gyrus fornicatus, but without any affection of the suprajacent marginal convolution.

In addition to these instances, in which the lesion was of a stationary character, some cases of tumour may be mentioned.

Dr. Handford has recorded a case of tumour occupying the greater part of the left paracentral lobule, with bulging of the gyrus fornicatus, which caused almost complete paralysis of the left leg, and eventually some weakness in the arm and side of the face also. Cutaneous sensation, tactile, and painful, and thermal, was greatly impaired over the whole of the left side. The impairment of sensation in this case was much greater than that which occurs from tumours in the Rolandic area.

A similar case has been reported by Ackerman. This was a case of tumour on the mesial surface of the left hemisphere, involving the greater part of the gyrus fornicatus and quadrate lobule. The brain was slightly softened immediately round the tumour, but both the convexity and base of the brain, as well as the centrum ovale and basal ganglia, were quite normal. The symptoms in this case were weakness of the right leg, followed by a similar condition of the arm, together with general hyperæsthesia. Under observation the patient fell into complete hemiplegia and analgesia, with conjugate deviation of the head and eyes to the left.

Muratoff reports a case of tumour in the mesial aspect of the left hemisphere, which largely destroyed the gyrus fornicatus, and was adherent to the corpus callosum. Above, it was limited by the calloso-marginal sulcus, and the paracentral lobule was intact. In this case there was some degree of weakness and awkwardness of the limbs on the right side, and touch and pain were abolished. At a later date it was noted that tactile sensibility was normal, but the sense of pain diminished. The author is of opinion that the affection of sensibility observed in this case was not the direct result of the focal lesion, but probably due to interruption of the associating fibres, and compression of the paracentral lobule.

But though it is necessary to be cautious in drawing conclusions from cases of tumour, the three above mentioned exhibit a remarkable agreement as regards the affection of sensation on the opposite side of the body when lesions of this character invade the falciform lobe. Taken with the symptoms observed in the two cases of stationary lesion, they lend at least considerable support to the experimental results obtained by Horsley, Schäfer, and myself.

That the motor centres of the Rolandic area are in intimate organic and functional relation with the sensory centres there can be no doubt, and they probably form a couple connected by associating fibres. Dr. Beevor shows that the falciform lobe is connected with the motor cortex by means of the fibres of the cingulum (2). It is probable that these fibres form the nexus between the Rolandic zone and the falciform lobe, and that it is through these, or similar connections, that irritative lesions of the motor zone are apt to excite eccentric sensations in the limbs, and that destructive lesions may produce temporary anæsthesia by causing dynamical disturbance of the centres of the falciform lobe.

According to the observations of Flechsig (8), with which those of Turner and myself coincide, the falciform lobe is the terminus of a system of cortici-petal fibres (System II.) which ascend from the optic thalamus, and are probably the direct continuation of the short longitudinal fibre-system of the tegment. We are of opinion, from the results of our experiments, that in these lie the true sensory paths. Though the fibres of the mesial fillet ascend to the Rolandic as well as to other cortical regions, there is no satisfactory evidence that they are concerned with the transmission of any of the forms of conscious sensation. The mesial fillet may be entirely degenerated without loss or impairment of cutaneous sensation (6).

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Lesions of the parietal lobe.—The parietal lobe includes the *superior* and *inferior* parietal lobules, the latter formed by the angular gyrus and supramarginal lobule. The superior parietal lobule, or upper extremity of the ascending parietal or post-central convolution, has already been considered as belonging to the Rolandic area, and will, therefore, not be specially considered further.

Lesions of the parietal lobe are frequently associated both with lesions of the Rolandic area and of the occipital lobe. Occasionally, however, the inferior parietal lobule is affected alone; and the question is then whether there are any symptoms specially related to lesions in this region capable of being accurately diagnosed. The corresponding region in the brain of the monkey constitutes the *pli courbe*, or angular gyrus, in which we distinguish an anterior and posterior limb. The anterior limb in man at its lower extremity forms a supramarginal gyrus.

There has been much difference of opinion in respect to the physiology of this region. I have found that electrical irritation in the monkey gives rise to very definite and constant effects; namely, to conjugate deviation of the eyes, and occasionally of the head, to the opposite side, with a direction upwards or downwards, according as the anterior or posterior limb is irritated. These results were also obtained by Luciani and Tamburini; but Schäfer appears to have observed them only on irritation of the posterior limb. Similar movements of the eyes to the opposite side, and with an upward or downward direction, are also excited by stimulation of various parts of the occipital lobe.

My hypothesis is, that these movements are the expression of subjective visual sensation; and that the regions from which they can be obtained by electrical stimulation are therefore related to the sense of vision.

My early experiments, as well as those subsequently made in conjunction with Professor Yeo, and more recently with Dr. Turner, have shown that, in the monkey, destruction of the angular gyrus causes total loss of vision of temporary duration in the opposite eye; whereas bilateral destruction causes temporary total blindness, which gives place to a peculiar impairment of vision of an enduring character; this I have interpreted as being more particularly due to loss of central vision. The experiments of Munk and Schäfer also show that monkeys after this bilateral lesion exhibit a want of precision in seizing small objects, though these authors interpret the symptoms differently from myself. On one point, however, our experiments are in harmony, namely, that destruction of the angular gyri does not give rise to motor paralysis either of the limbs or the ocular muscles. In particular there is no ptosis; and on this subject we may here consider the views advanced by Landouzy (12), Grasset (9), and Wernicke.

These observers are of opinion that clinical facts indicate that the *pli courbe*, or angular gyrus, is the cortical centre for the levator palpebræ superioris, as well as for the conjugate movements of the eyes to the opposite side; and they adduce cases of unilateral ptosis, and conjugate deviation of the eyes to the side of lesion, in which the angular gyrus has been found to be the seat of lesion.

Thus Chauffard has recorded a case of word-blindness and deafness due to lesion of the left angular and supramarginal gyrus in which there was some degree of ptosis of the right eye.

Lemoine has reported a case of slight paresis of the left limb, with ptosis and slight external strabismus of the right eye. The patient died some years after of apoplexy and left hemiplegia. In addition to the lesions which caused the left hemiplegia there was in the left hemisphere an old lesion exactly occupying the angular gyrus, and extending towards the parallel and intra-parietal fissures. There was said to have been no alteration in the pons, medulla, or oculo-motor nerves.

Surmont has reported a case of ptosis of the left eye, and rotation of the head to the right, without paralysis of the limbs. In the right hemisphere there was found a yellow softening occupying the upper half of the posterior third of the first temporal convolution and the lower half of the inferior parietal lobule, extending slightly into the ascending parietal convolution.

Grasset (8) has reported a case of ptosis of the left eyelid, in which after death was found a patch of exudation, with intense congestion of the cortical substance, at the upper extremity of the parallel fissure in the immediate neighbourhood of the right angular gyrus. There were, however, in this case, as Charcot and Pitres remark, signs of diffuse meningitis on the convexity of both hemispheres.

In a case of conjugate deviation of the eyes to the right, reported by Wernicke, in addition to lesion of the medullary fibres of the inferior parietal lobule in the right hemisphere, there was a lesion also in the right half of the pons.

In the great majority of the cases in which conjugate deviation of the eyes has been observed, there has also been hemiplegia; and the lesions, if they involved the inferior parietal lobule, invaded the motor region also; while in some cases there was no lesion whatever in the angular gyrus or in its neighbourhood.

On the other hand, a large number of cases are on record in which, notwithstanding extensive lesions of the angular gyrus, no ptosis was observed.

Surmont has found only 11 cases of blepharoptosis, against 50 in which this symptom did not occur.

In a collection of strictly cortical lesions implicating the angular gyrus (analysed for me by Dr. Ewens), in 30, in which the angular gyrus was affected along with the Rolandic zone, there was no ptosis or conjugate deviation of the eyes; in 16 cases of lesion of the parietal lobe, in 6 of which the angular gyrus was alone affected, ptosis occurred only in 2; while in 13 cases of lesion of the parieto-temporal region, in 10 of which the angular and upper temporal gyri were affected, only one showed conjugate deviation of the eyes. It is obvious, from this analysis, that neither ptosis nor conjugate deviation of the eyes is a common symptom in connection with lesions of the inferior parietal lobule; and that clinical facts do not justify the assumption of any causal connection between them.

The facts of experiment, however, would lead us to expect that irritation of the inferior parietal lobule might cause conjugate deviation of the eyes to the opposite side. And a case in confirmation of this hypothesis has been recorded by Thompson. This was a case of fracture of the skull in the left parietal region—cured by trephining—in which there was marked conjugate deviation of the eyes to the right. On the other hand, a similar symptom was produced by a patch of tuberculous meningitis over the upper part of the middle frontal convolution, in a case reported by Chouppe.

Wernicke's hypothesis that double lesion of the angular gyrus causes a pseudo-nuclear ophthalmoplegia is supported neither by experimental physiology nor by clinical evidence of any value.

The only constant symptom which has been established in connection with lesion of the inferior parietal lobule is the occurrence of word-blindness when the lesion is in the angular gyrus of the left hemisphere. This may, or may not, be associated with right hemianopsy. Many instances of this affection are on record. One of unusual interest has been reported by Macewen. This was a case of traumatic lesion of the left angular and supramarginal gyrus causing word-blindness and mental disturbance, but no other symptoms. The patient recovered after trephining. In a case recorded by Henschen (10), softening of the left angular gyrus was associated with word-blindness. A similar condition existed in Chauffard's case together with slight ptosis of the right eyelid. A large number of similar instances might be quoted (*vide* Starr, 18), all bearing out the fact that lesions of the angular gyrus and its neighbourhood in the left hemisphere cause, often without any paralytic symptoms whatever, the special form of sensory aphasia called word-blindness. The pathology of word-blindness and its relation to lesion of the angular gyrus is chiefly a matter of speculation. My own view, founded on the effects of unilateral and bilateral lesion of this gyrus in monkeys, is that each angular gyrus is in relation with the whole visual field of both sides, but especially with the opposite. Sir William Gowers also is of opinion that in front of the occipital lobe—the half vision centre—there is a higher visual centre in which the half fields are combined, and the whole of the opposite visual field is represented.

The bilateral relations of each angular gyrus are sufficient to explain the absence of any noteworthy affection of vision when the lesion is only on one side. And the fact that visual ideation, more particularly in reference to the association of written symbols with their meaning—that is, word-vision—is specially impaired by lesion of the left angular gyrus, seems to show that visual re-presentation is in this respect like auditory re-presentation (see word-deafness, p. 319); and that lesion of the area of highest vision may paralyse visual re-presentation, while the simpler function of visual sensation or presentation, like that of audition, may not be appreciably affected.

There is comparatively little clinical evidence in favour of the occurrence of crossed amblyopia from unilateral cortical lesion. But Dr. Sharkey has recorded a case of this kind in which the angular gyrus of the opposite hemisphere was for the most part atrophied. And cases of crossed amblyopia from unilateral organic cerebral lesion have als been recorded by Féré, Gowers, and myself (6). The facts of hysterical hemianæsthesia, in which vision is lost, or impaired, in the eye on the anæsthetic side, also postulate the existence in the opposite hemisphere of a centre related to the whole visual field of the opposite side, as well as to some extent on the same side.

Dr. Hughes Bennett has shown that irritative lesions of the angular gyrus give rise occasionally to optical illusions or flashes of light followed by temporary amblyopia.

Bilateral destruction of the angular gyrus, which alone, according to my experimental results, should cause permanent loss or profound impairment of vision, is a rare occurrence. I know of only one case on record — Shaw's case — and the lesion (atrophic softening) invaded not only the angular gyri, but also the superior temporal convolutions on both sides. The patient, who suffered also from mental aberration, was totally blind as well as deaf.

Not infrequently, where the lesion of the angular gyrus and adjacent portions of the temporo-sphenoidal and occipital lobes has been of the nature of tumour, or has invaded also the medullary substance, there has been observed, in addition to word-blindness, hemianopsy, or hemianopsy associated with a greater or less degree of impairment of tactile and muscular sensibility of the opposite side. Many cases of this kind might be cited (Henschen, 11). In all of them no doubt the optic radiations, together with the posterior part of the internal capsule, were involved to a greater or less extent; for an equal number of cases might be quoted in which similar lesions, not implicating the optic radiations or internal capsule, did not cause hemianopsy, or any apparent affection of sensation.

Summary.—Lesions of the inferior parietal lobule are diverse in their symptomatology. They may be entirely latent, or associated with ptosis, hemianopsy, or impairment of cutaneous and muscular sensibility of the opposite side.

They can only be diagnosed with certainty when situated in the left hemisphere, in which case the pathognomonic symptom is the form of sensory aphasia termed "word-blindness," or inability to comprehend the meaning of written language.

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Lesions of the occipital lobe.—As I have already stated, electrical irritation of the cortex of the occipital lobe in monkeys gives rise to movements of the eyes, similar to those which I first described on irritation of the angular gyrus. Professor Schäfer obtained upward movements on stimulation of the under surface of the occipital lobe and lower part of the mesial surface, but simply lateral movements of the eyes on excitation of the convex aspect of the occipital lobe and margin of the longitudinal fissure. These results have been considered by Schäfer as indicating a special relation of the different portions of the visual field to respective regions of the occipital cortex. Whether this be so in reality has not been proved; and the results of destructive lesions show that the relation, if it exist at all, cannot be regarded as an exclusive one, inasmuch as the greater portion of both occipital lobes may be removed without causing more than transient visual disturbances, and without producing permanent blindness in any part of the visual field. Unless the lesions are such as to implicate the parieto-occipital fissure, and thus in all probability to cut off the whole of the optic radiations, monkeys at least continue to see; and Schäfer's experiments show that in order to cause complete loss of vision, not only must the occipital lobes, as such, be cut off, but the lesion must extend well forward into the occipito-temporal convolutions. I have shown (2) that a transverse incision a few millimetres in depth on the under surface of the occipital region, anterior to the lower extremity of the parieto-occipital fissure, without the infliction of any injury upon the angular gyrus or occipital lobe, can cause persistent hemianopsy; and my view is that by removal of the occipital lobe in this plane the whole of the optic radiations are severed. The exact destination of the optic radiations is a problem which has been much discussed, but that they distribute themselves in the occipito-parietal region of the brain there can be no doubt; for when, not the occipital lobe only is destroyed, but also the cortex of the angular gyrus, complete and persistent hemianopsy ensues if the lesion is unilateral, and total blindness if the lesion is bilateral. Practically the same result is obtained by section in the plane of the parieto-occipital fissure, by which the whole of the optic radiations are severed. In harmony with the fact that in monkeys a large proportion of the occipital lobe may be destroyed without producing obvious disturbance of vision, we find many clinical cases in which the occipital lobe has been the seat of lesion, unilateral and bilateral, without any impairment of vision. Lesions of the occipital lobe may be, in fact, entirely latent. Such lesions have been found on the superior, lateral, and inferior aspect, as well as at the apex of the occipital lobe, or even of the mesial aspect or cuneus. Thus Sir William Gowers has reported a case of tumour of the right occipital region involving the first and second occipital convolutions, the superior and inferior parietal lobules, together with the cuneus and half of the precuneus, in which careful investigation failed to discover any indications of hemianopsy. Brill has reported a case of thrombotic softening of the mesial aspect of the left occipital lobe, including the cortex bordering on the calcarine fissure, the upper edge of the lingual gyrus, and the lower half of the cuneus, in which there was no absolute defect in the visual field, though the patient had diplopia and was colour-blind as regards green.

In the majority of cases in which hemianopsy has been found in association with lesions of the occipital lobe, the lesion has either been of the nature of a tumour, cyst, or abscess, or, if invading the cortex, has also extended into the optic radiations. In many the lesions have been multiple and not confined to the occipital region; and in the great majority also, in addition to hemianopsy, there has been hemiplegia, hemianæsthesia, aphasia, or other symptom of implication of the internal capsule or cortical centres beyond.

The clinical facts, however, seem to show that, so far as lesions apparently cortical are concerned, hemianopsy is most frequently associated with those affecting the mesial aspect of the occipital lobe.

Seguin, Nothnagel, and others place the visual centre more especially in the cuneus and its neighbourhood; Henschen and Vialet restrict it to the calcarine fissure; while Wilbrand regards the apex of the occipital lobe as the part exclusively related to vision. If a complete homology exists between the occipital lobe of the monkey and man, one may argue that each of these views is too exclusive. But, apart from this, it is a question how far an examination of the recorded cases justifies the restriction of the visual centre to one particular portion of the occipital lobe.

Most of the apparently cortical lesions of the mesial aspect have been cases of thrombotic occlusion of the posterior cerebral artery, which supplies not the cortex only, but also the subjacent optic radiations, separated from the mesial wall only by the posterior cornu of the lateral ventricle, which, therefore, may easily suffer at the same time as the cortex.

Examination of 23 cases which have been quoted by Henschen (*op. cit.* p. 319) as purely cortical lesions of the mesial aspect, reveals the fact that in at least 6 of them (namely, those of Sänger, Curschmann, Huguenin, Henschen's case 21, Reinhardt's case 9, and Wilbrand) the optic radiations were also involved; in those of Leegard, Féré, and Noyes there was no investigation into the condition of the optic radiations; while in 14 there was, in addition to hemianopsy, also some degree of hemiplegia, or hemiplegia with hemianæsthesia.

Of three cases of hemianopsy, reported by Vialet, attributed to purely cortical lesion, the first was one of softening occupying principally the internal perpendicular fissure, but extending also into the calcarine fissure. At the same time the cortex of the cuneus was atrophied in its anterior fourth. The lesion, however, was not strictly cortical, but penetrated to the depth of a few millimetres into the zone of the optic radiations. The second case was one of right hemianopsy associated with a lesion which had destroyed the cuneus, extended to the lips of the calcarine fissure, and slightly affected the white substance of the lingual lobule. The internal capsule was stated to have been intact, and yet in this case there was, at the beginning of the illness, turning of the head to the left and rigidity of the right limbs; and marked defect in the sense of position of the right arm persisted. In the third case there was right hemianopsy together with word-blindness. The lesion in this case was recent softening of the whole of the inferior parietal lobule and *pli courbe*, together with the *plaques jaunes* occupying the lingual lobule, the fusiform lobe, the cuneus and the apex of the occipital lobe, as well as the splenium of the corpus callosum. The optic radiations were, however, also found softened in this case, though this was attributed to recent lesion.

It is evident from this analysis that in the great majority of the so-called cortical lesions of the mesial aspect of the occipital lobe the lesion extended more deeply than was apparent on the surface; and even when the optic radiations were not demonstrably affected, they were in all probability directly implicated along with the internal capsule, as otherwise it would be impossible to explain the hemiplegia and hemianæsthesia with which the hemianopsy was so often associated.

The only case, in fact, in which all complications seem to have been excluded is the case of Nordensen-Henschen, in which left hemianopsy was associated with cortical softening of the right calcarine and upper part of the hippocampal fissure. Microscopical examination revealed degeneration, but apparently no primary softening of the optic radiations. If we assume that the degeneration of the optic radiations was merely secondary to the cortical lesion, this case might be regarded as a proof that in man the cortex of the calcarine fissure is pre-eminently the visual centre. But in my opinion it is not to be so regarded without further investigation and confirmation. For there can be no doubt from the researches of Vialet and von Monakow, as well as those of Turner and myself, that the optic radiations have a much more extensive distribution than the walls of the calcarine fissure. A considerable proportion, the middle stratum, pass to the calcarine fissure; but an inferior layer can be traced to the lingual and fusiform lobules, and a superior layer to the angular gyrus and convex aspect of the occipital lobe. The visual area must embrace, therefore, not merely the calcarine fissure, but the whole of the occipito-angular region (10). Whether there be any specific differentiation of function corresponding to the tripartite distribution of the optic radiations is a question on which little can be definitely stated.

An examination of the clinical facts supports the view that the macula lutea is innervated from both cerebral hemispheres. The dividing line between the sensitive and blind halves of the retina in homonymous hemianopsy of cerebral origin probably never passes through the fixation point. Round this there is always an area of greater or less extent of clear central vision. I have only once seen the dividing line pass through the fixation point, and this was in a case of hemianopsy from lesion of the optic tract itself. It is frequently stated, however, in cases of cerebral hemianopsy that the dividing line had passed through the fixation point; but such statements are to be accepted with great caution, as mistakes in perimetric investigations are easily made.

I have adduced some evidence in favour of the view that the macula lutea is bilaterally represented in the angular gyrus. Henschen, however, is of opinion that the macula is represented in the anterior portion of the calcarine fissure. But the only evidence which he adduces in favour of his hypothesis is the fact that central vision continued good in a case in which the whole of the right occipital lobe and the apex of the left were destroyed—indicating, therefore, that the macula was not innervated from the posterior extremity of the calcarine fissure.

Occasionally, instead of complete blindness of one-half of the visual field, sector or quadrant-like defects are found in the upper or lower half; and it has been argued by Munk and Schäfer, on the basis of their experimental researches, that the different parts of the retina are projected on corresponding portions of the visual cortex. The evidence of experimental lesions on this point is, however, far from satisfactory, and the clinical evidence is no more so. Henschen, however, believes that it points to the conclusion that the upper lip of the calcarine fissure represents the dorsal retinal quadrant, and the lower lip the ventral quadrant. This is opposed to the conclusions of Munk, from which also those of Schäfer materially differ. Further investigation is therefore necessary before we can regard any of these points as definitely determined.

The same may be said with reference to the hypothesis advanced by some observers that there is a cortical centre for colour as distinct from light-perception. In some cases there has been observed a homonymous hemichromatopsy, while the perception of light and form was not obviously, or correspondingly, impaired. But Mackay has furnished strong grounds for regarding such cases as merely varying degrees of essentially one affection; for careful investigation has always revealed not loss of colour perception only, but also distinct impairment of light and form perception; and, as the lesion becomes more pronounced, hemichromatopsy passes into complete hemianopsy.

In cerebral hemianopsy the pupils react in the usual manner when a pencil of light is thrown on the blind halves of the retinæ. The non-occurrence of the pupil reaction when the retinæ are so tested (Wernicke's hemiopic pupillary reaction) is significant of lesion of the optic tract. (On this subject see vol. vi. p. 765.)

Summary.—(i.) Lesions of the occipital lobe may be entirely latent.

(ii.) Homonymous hemianopsy results from destructive lesions of the occipital lobe and optic radiations. This is often associated with a greater or less degree of hemiplegia and hemianæsthesia, from implication or the internal capsule. When the lesion affects also the cortex of the angular gyrus, right hemianopsy is generally associated with word-blindness.

(iii.) Uncomplicated hemianopsy, occurring suddenly with symptoms of vascular obstruction, is indicative, in all probability, of lesion in the calcarine fissure and its neighbourhood.

(iv.) In cerebral hemianopsy central vision is unimpaired, and the reaction of the pupils is normal.

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LESIONS OF THE TEMPORAL LOBE.—Owing to the community of vascular supply between the parietal, occipital, and temporal lobes, the last is not infrequently implicated in lesions due to vascular disease affecting either the parietal or occipital lobe, or both together. The middle cerebral artery, which supplies the Rolandic area, also supplies the angular gyrus and the convex aspect of the temporal lobe; namely, the first and second temporal convolutions. The inferior and inner aspect of the temporal lobe is supplied by the posterior cerebral artery, which is also distributed to the cuneus and lingual gyrus on the internal aspect of the occipital lobe. The temporal lobe is specially connected with the outermost fibres of the foot of the crus cerebri, which pass up into the internal capsule, and then bend outwards and downwards to this region. In cases of lesion of the temporal convolutions these fibres have been found by Déjerine to degenerate downwards as far as the upper region of the pons. This has been confirmed by Turner and myself after lesion of the first temporal gyrus.

Lesions of the temporal lobe, of whatsoever character, may be entirely latent. There are so many such cases on record that it is unnecessary to allude to them in detail. This latency is more particularly notable when the lesions involve the right lobe, the explanation of which is to be found in the greater preponderance of the left hemisphere in speech processes.

One of the most important indications of lesion of the left temporal lobe is the occurrence of the speech defect termed "word-deafness." This signifies the inability to interpret the meaning of articulate sounds. It may exist independently of word-blindness, though, owing to conjoint lesion of the inferior parietal and temporal lobes, the two conditions are often associated together. In all cases in which word-deafness has been a prominent symptom a lesion has been found in the superior temporal convolution. Thus Seppilli (20) found that in every one of seventeen cases there was lesion of the superior temporal convolution; and in twelve in the second or middle convolution also. Of twenty-five recorded cases, analysed for me by Dr. Ewens in 1890, ten were due to lesion of the temporal lobe alone. In seven of these the first temporal was particularly affected; but in the remaining three the exact limits of the lesion were not stated. Eight were due to lesions implicating the angular gyrus, as well as the upper temporo-sphenoidal. Six were due to lesions invading the upper temporal gyrus and adjoining portions of the occipital and parietal lobes; and in one it was stated that the angular gyrus was affected alone. In this case, however, both word-blindness and word-deafness were present. This was the only case in which there was no obvious lesion of the superior temporal convolution, but since then other cases have been put on record. Thus Mader reports the case of a patient, aged fifty, who was suddenly seized with disturbance of speech and complete word-deafness. After death a yellowish white softening was found affecting the left superior and middle temporal convolutions. A similar case, due to lesion of the posterior part of the first temporal convolution and adjoining portion of the inferior parietal lobule, has been recorded by Fraser.

The relation of word-deafness to lesion of the left hemisphere appears to follow the same rule as obtains in reference to aphasia and lesion of the speech centre of the left side. Thus Seppilli (21) has recorded in a left-handed man a case of atrophy and sclerosis of the first and second temporal gyri in the left hemisphere without any disturbance of speech or hearing; and a case of a large tumour invading the whole of the left temporal lobe, without affection of speech or hearing, in a patient supposed to be left-handed has been reported by Westphal (25).

The facts of word-deafness are explicable on the hypothesis that the region in which the lesions are habitually found constitutes the cortical seat of the perception and registration of auditory impressions. My own experiments on monkeys, as well as similar experiments by Munk, and by Luciani and Tamburini on dogs, indicate that in this region the centre of auditory perception is situated. I have found, in particular, that electrical irritation of the superior temporal gyrus causes sudden movements of the ear, head, and eyes, such as are indicative of perception of sound in the opposite ear; and that destruction of this gyrus on both sides causes total deafness, or loss of auditory perception.

An animal in which the superior temporal gyri were almost completely destroyed on both sides at first absolutely failed to respond to any of the sounds which formerly excited active reaction, and which invariably attracted the attention of normal monkeys. After some weeks it seemed to be in some degree sensible to sonorous vibrations, but it seemed never to recognise the sounds which formerly were of significance to it. The obscure sensibility to auditory impressions, which was observed in this animal, might have been due either to imperfect removal of the auditory centres, or perhaps to the functional activity of the lower centres. Professor Schäfer has been unable to confirm my conclusions with reference to the relation of this convolution to the sense of hearing; but the facts of pathology, not only in regard to word-deafness, but also to actual deafness resulting from bilateral lesions in this region, afford ample confirmation of my results.

Owing to the bilateral relations of the auditory centres, individuals who are word-deaf are not otherwise, as a rule, deaf to sounds. The probability, however, is that there may be transient impairment of hearing in the opposite ear.

There are several cases on record in which bilateral lesion of the superior temporal convolution and neighbourhood caused complete deafness. One of the most carefully observed cases of this kind has been recorded by Wernicke and Friedländer. A woman, aged forty-three, who had never suffered from deafness or affection of vision, was attacked on 22nd June 1883 with right hemiplegia and aphasia. She remained in the hospital until 4th August. At this time she could speak, but she spoke unintelligibly and was sometimes believed to be intoxicated. She not only could not make herself understood, but she could not understand what was said to her. She was received into the hospital again on 10th September, with slight paresis of the left arm. The right hemiplegia had entirely disappeared. The patient was looked upon as insane; she was absolutely deaf, and could not be communicated with. She died on 21st October. An extensive lesion was found invading the superior temporal convolution on both sides. The rest of the brain exhibited no abnormality, nor were there any conditions leading to increase of the intracranial pressure or secondary affection of the cranial nerves. The patient had previously enjoyed excellent health, her total deafness occurring suddenly in connection with the other indications of cerebral disease. The result of the examination of the ears was practically quite negative, a slight dry catarrh was found, but locally nothing to account for deafness.

Shaw has recorded a case of a woman, aged thirty-four, who, two months before her admission into his asylum, lost power in the right arm, and soon after had a sudden apoplectic seizure, resulting in loss of speech and deafness. On admission she was found to be perfectly deaf and blind. She had occasional fits, and ultimately died of pneumonia a year after her admission. There was complete atrophy of the angular gyri and superior temporal convolutions in both hemispheres. The optic nerves exhibited increase of the connective tissue, but the other cranial nerves were normal in appearance. It may perhaps be questioned whether the blindness were due to the lesion of the angular gyri alone, or to secondary changes in the optic nerves; but the sudden onset of deafness in this case, coincident with symptoms of cerebral lesion, and the condition of the brain, point to the destruction of the superior temporal convolutions as its cause.

A third case of a similar nature has been recorded by Mills (14). The patient was a woman, aged forty-six, who fifteen years before her death had an apoplectic attack which left her word-deaf, but not paralysed. She could hear sounds, though she could not understand what was said to her. Six years later she had a second apoplectic attack, causing partial left hemiplegia, together with total deafness for sounds as well as words. She was examined by Mills a few days before her death, which occurred from asthenia and emaciation. In the left hemisphere the posterior two-thirds of the first temporal convolution were reduced to a thin strip; and at the posterior quarter of the second temporal convolution was a cavity, evidently the remains of an old embolic softening. The lobe was otherwise normal. There was, however, some degree of atrophy of the lower portion of the central convolutions and hinder part of the first frontal. In the right hemisphere was an old hæmorrhagic cyst destroying the first and second temporal convolutions, the insula and the lower end of the central gyri, together with the lenticular nucleus and external capsule. The auditory nerves were atrophied, and the striæ acusticæ, usually so easily seen, could not be made out with the naked eye.

Even in cases of unilateral lesion of the superior temporal convolution, some observers have noted deafness in the opposite ear; but the cases are not altogether free from doubt, as the condition of the ears does not appear to have been quite satisfactorily examined. Thus Kaufmann has reported a case of obliteration of the chief branch of the right Sylvian artery, causing softening of the first and second temporal gyri, together with atrophy of the lower end of the posterior central and supramarginal gyri. The symptoms in this case were left hemiplegia without aphasia, and total deafness in the left ear, though the patient was said to have heard well before the attack. Ferguson has recorded a case in which there was already long-standing deafness in the right ear from otitis media. Two years before death convulsive movements occurred on the left side accompanied by an auditory aura, and followed ultimately by entire loss of hearing in the left ear. The necropsy revealed the existence of a tumour affecting the first and second temporal convolutions in the right hemisphere, destroying the first entirely, and the second partially.

Irritative lesions in this region occasionally cause, as in the case last mentioned, an auditory aura, whether the lesion be in the right or the left hemisphere. Gowers has recorded two cases of this nature: in the one, a tumour, of which the oldest part was beneath the superior temporal convolution, caused convulsions beginning with an auditory aura referred to the opposite ear; in the other a tumour affecting the superior temporal gyrus caused unilateral convulsions preceded by a loud noise as of machinery.

Dr. Hughes Bennett also has reported several cases of auditory sensory discharges followed by temporary loss of hearing in the opposite ear or in both. Thus, a woman, subject to epileptic attacks preceded by a loud noise like the ringing of a bell in the left ear, became temporarily deaf in both ears after each attack. Both ears were defective in hearing, but the left more so. And in a case of abscess in the right temporal obe, due to otitis media, recently under my care the prominent symptom which indicated the seat of the lesion was the occurrence of auditory hallucinations towards the left, so vivid that when the patient was under examination he would occasionally suddenly turn to the left, pointing and saying, "Didn't you hear that?" The abscess was evacuated, but the patient succumbed.

Of important bearing on the question of the position of the auditory centre are the facts relating to the condition of the brain in deaf-mutes and in cases of deafness of long-standing. Mills (14) has described the brain of a woman, who was deaf for thirty years, which, otherwise normal, exhibited extensive atrophy of both superior temporal gyri, particularly of the left. And Sir William Broadbent has described the brain of a deaf-mute in which, in addition to some defect of the annectant convolutions and supramarginal lobule, there was atrophy of both superior temporal gyri, especially of the left.

It has been found by von Monakow, Zacher, and Déjerine, and confirmed by Turner and myself, that lesions of the first temporal gyrus lead to degeneration downwards in the outer portion of the pes cruris traceable as far as the upper pontine region, but not affecting the so-called central auditory tract.

The temporal lobe is a frequent seat of abscess in connection with disease of the middle ear (*vide* subsequent article). When the left temporal lobe is so affected the diagnosis is frequently rendered easy by the occurrence of word-deafness and disturbance of speech. When the abscess is situated in the right temporal lobe the diagnosis is more difficult, and may be impossible unless there be some degree of hemiplegia, hemianæsthesia, hemianopsy, or pressure on some of the cranial nerves, of which the third is specially liable to suffer. Apart from such conditions the diagnosis cannot be made with certainty, as the whole of the temporal lobe, even of the left side, may be destroyed without obvious symptoms, provided the superior temporal gyrus is unaffected.

A case has been described, however, by Mills and M'Connell which they consider indicative of the existence of a "naming" centre in the third temporal gyrus. The case was that of a woman, aged 41, whose symptoms began with vertigo, followed by word-blindness and right hemianopsy. She could not name objects either by sight or touch, though she understood what they were and what were their uses. Right hemiplegia set in, followed by death in a state of coma. The necropsy revealed a glioma involving the posterior part of the third temporal convolution, and extending slightly into neighbouring parts. The postulation of a "naming" centre on facts such as these appears to me to involve far greater difficulties than to explain them by indirect or dynamic disturbance of the centres of visual ideation.

The facts of comparative anatomy, as well as of physiological experiment, indicate that at the lower extremity of the temporal lobe, more particularly in the hippocampal lobule, or anterior extremity of the hippocampal gyrus, to which the external root of the olfactory tract is traceable, is situated the cortical centre of smell. Electrical irritation of this lobule causes movements of the nostril, particularly on the same side, which have the appearance of subjective olfactory sensation. And certain experiments, which I have elsewhere related in detail (7), show that destruction of this region in both hemispheres renders the animal incapable of perceiving odours, or even of recognising tastes, which under normal circumstances excite antipathy and disgust. My hypothesis is that in this portion of the cortex are situated not only the principal centres of smell, but also of taste; though the limits of each have not as yet been defined with accuracy. Some clinical cases of lesions of the temporal lobe have been recorded which are in favour of this view, though not sufficiently precise to prove it.

Churton and Griffiths report a case in which smell was impaired on the side of lesion, by a tumour which caused erosion of the uncus, and which did not appear, directly at least, to have affected the olfactory tract; and several cases have been recorded in which olfactory auræ, or crude sensations of smell, have been observed in connection with lesions implicating this part of the temporal lobe.

M'Lane Hamilton reports a case of this kind due to meningitis involving the inferior and anterior extremity of the temporal lobe, without implication of the olfactory tract.

Jackson and Beevor have described a case of olfactory aura occurring in connection with a tumour of the anterior end of the right temporal lobe involving the nucleus amygdalæ and hippocampal lobule. A similar case has been recorded by West, and another by Sander. In the latter case epileptiform convulsions were preceded by an unpleasant odour. The lesion was a tumour, which involved the anterior extremity of the temporal lobe, but the olfactory tract was also directly involved; and several other instances of a similar character might be referred to. Owing, however, to the implication of the olfactory tract, some of them at least might be ascribed to direct irritation of this structure; but in others this was not evident, and the aura probably depended upon irritation of the olfactory centre itself.

A case in which, in addition to subjective olfaction, gustatory sensation seems to have been excited, has been related by James Anderson. This occurred in connection with a tumour affecting the left temporal lobe, but the lesion was not of a character to allow of any precise conclusions being founded on it as to the position of the gustatory centres. Apart from their physiological bearing, these cases are of importance in a diagnostic point of view, and indicate that lesions which affect the sense of smell have their seat at the lower and anterior extremity of the temporal lobe.

Summary.—(i.) Lesions of the temporal lobe may be entirely latent.

(ii.) Word-deafness is indicative of destructive lesion involving the superior temporal gyrus of the left hemisphere.

(iii.) Subjective sensations of smell and taste are indicative of irritative lesions involving the lower extremity of the temporal lobe.

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Lesions of the corpus callosum.—The commonly received view of the constitution of the corpus callosum is that it forms a great commissure connecting identical points of the two hemispheres with each other. The parts that are not connected together by the corpus callosum, namely, the hippocampal lobules, are, according to Flower, connected by the anterior commissure, which thus forms a supplement to the corpus callosum, and varies inversely with it. This view has been contested by Hamilton, who holds that the corpus callosum is not a commissure in the ordinary sense, but rather a decussation of certain cortical fibres which do not decussate lower down. He does not, however, adopt Foville's view that the corpus callosum is a decussation of the peduncular fibres. It has been maintained by Schnopfhagen, and also by Sachs, that the corpus callosum is a system of associating fibres connecting, not homologous parts of the hemispheres, but regions quite distinct in situation and in function.

The experimental and histological investigations of Muratoff (10) are, however, in favour of the commonly adopted view as to the commissural function of the corpus callosum. Thus he found, after excision of different portions of the motor cortex, that degenerated fibres could be traced from the excised area across the corpus callosum as far as the cortex of the identical contra-lateral area,—the number of degenerated fibres being directly proportional to the extent of the cortical lesion, and always limited to a particular region of the corpus callosum, according to the site of the lesion. The degeneration was always more extensive when an identical lesion was made on both sides than on one side only. On section of the corpus callosum, complete or partial (12), he found degenerated fibres traceable to the cortex on both sides quite identical on the two sides, and lying in a strictly defined field corresponding to the extent and site of the lesion of the corpus callosum. He also found that a simultaneous destruction of the cortex and section of the corpus phenomena above described continue as before, but in a more exaggerated form. It is unnecessary to describe them again.

At a yet later stage progression sometimes becomes almost impossible, the patient lies in bed with the limbs rigid, every attempt at movement bringing on more violent contractions of opposing muscles, and locking the limb in a tetanoid spasm. The prevailing muscles, notably the muscles of the calf, get into a state of permanent contraction, and ultimately become shortened; and upon this follow deformities of the nature of talipes; and, if the patient is still able to get out of bed and to move about, the difficulty of locomotion is greatly increased by these deformities. The disease may at the same time creep insidiously upwards, involving the muscles of the trunk and arms, even extending into the bulb and resulting in bulbar paralysis; but before this is reached it passes out of the group of pure lateral sclerosis into that of amyotrophic lateral sclerosis.

So after many years it may happen that death results from extension of the sclerotic process to the vital centres; but the fatal result is often due to other causes, as for example to an intercurrent pneumonia or pleurisy, to renal disease, or to vascular degeneration. The mere sclerosis of the lateral columns does not of itself suffice to induce death.

Causation.—The causes of the condition are very imperfectly known. It begins most commonly in adults between the ages of twenty and forty. It affects the sexes almost equally. I have been impressed with the importance of excessive muscular effort in some cases, and have had some reason to think that sexual excesses, particularly in the form of masturbation, may have had to do with its origin. Various infective processes have been regarded as causes. At the head of the list of these comes syphilis, followed, at a long interval, by acute infective fevers and by lead poisoning. With regard to hereditary influences it is difficult to speak with certainty; but observations in some measure confirm the conclusion, to which general principles would lead us, that it must be more common in neurotic families.

Diagnosis.—1. *From hysteria.*—The problem is often extremely difficult in the early stages of lateral sclerosis, for hysteria may closely mimic its symptoms, and we are occasionally compelled to suspend our judgment until repeated observations and the evolution of the symptoms may supply us with facts. The chief indications on which I rely in hysteria are the suddenness of development, and the occasional temporary disappearance of symptoms. I have never known these features in pure lateral sclerosis. Next to them are the indications derived from the presence or absence of a hysterical constitution. And, third, is to be ranked the existence of unmistakably hysterical phenomena of other kinds—say involving the sensory functions, the fields of vision, or other symptoms which are proper to hysteria, and foreign to lateral sclerosis. Amongst these special attention should be directed to the gait, the knee-jerk, and the precise quality of the ankle clonus, if it be present.

The gait in hysteria is apt to differ from that proper to lateral sclerosis in that the characteristic pure spastic toe-scraping movement is replaced by a gait in which the spastic element is, so to speak, overdone—the whole limb becoming rigid, and the heel as well as the toe being frequently scraped along the ground. Moreover, there is an almost indescribable "functional" appearance about all the movements; all difficulties are exaggerated by the patient, and the gait as a rule becomes worse when its features are being studied.

As to the knee-jerk, it may be much exaggerated in functional cases, but it is apt to be attended by a jerk of the whole mass of the erector spinæ, which I have never seen in a pure lateral sclerosis. Moreover, in organic spastic disease the knee clonus is often present, and although it is conceivable that such a phenomenon might be simulated in hysteria, I am not aware of any case in which it has actually been observed.

Attention should also be devoted to the precise character of the ankle clonus, if present; for in hysteria its features often differ from those proper to lateral sclerosis. While in hysteria we occasionally meet with a condition which can scarcely be distinguished from the ankle clonus of organic disease, we more frequently find what may be called a pseudo-clonus marked by the following features: (*a*) its mode of onset—instead of beginning with a movement of extension it begins with a dorsiflexion of the foot; (*b*) its course and persistency—it is ill-sustained, and instead of being capable of almost indefinite prolongation so long as the calf muscles are kept on the stretch by steady upward pressure upon the ball of the foot, it gradually and often speedily ceases.

The positive diagnosis of true lateral sclerosis thus turns mainly upon the persistence, continuity, and gradual progress of the symptoms, and its exclusion upon the presence of symptoms proper to hysteria.

2. *From secondary lateral sclerosis.*—I have met with many cases in which secondary disease existed, and yet in which the history of primary disease was far from distinct. Caries of the vertebræ, with little evidence of Potts' curvature, transverse myelitis of limited extent and of short duration, hæmorrhages, softenings, and tumours of the cord may be causes which are difficult to detect, or of which the patient may give a very imperfect history—the case, perhaps, coming under observation after the spastic phenomena have been long well established. But in the great majority of cases the preceding disease is perfectly manifest—and, even if indistinct, can be made out by careful examination and by a study of the history of the case.

3. *From multiple sclerosis.*—A number of cases which during life had shown symptoms closely resembling those of lateral sclerosis have been found on autopsy to present the multiple changes of insular disease. But it is not so much in the advanced stages as at an earlier period of the malady that the conditions are apt to be confounded. It rarely happens that a case of insular sclerosis, if somewhat advanced, does not betray its real nature by the superaddition to spastic phenomena of characteristic features proper to itself, such as the nystagmus, the optic atrophy, the

staccato speech, and the so-called "intention tremors," which I venture to suggest would be better designated "action tremors."

4. *From spastic paraplegia of cerebral origin.*—The diagnosis from this form of cerebral disease may be difficult; but in the case of children it should always be kept in view that spastic paralyses are often due to a cerebral lesion. Much light may be thrown upon the problem by the history of the onset of the malady, the evidence of intra-cranial damage, coma, or general convulsions followed by paraplegia. I do not deny that in childhood lateral sclerosis may occur, but cerebral lesions, whether pre-natal, natal, or post-natal, are much more common. At an early stage of the disease in adults, when the phenomena may be mostly confined to one limb, it may be confounded with post-hemiplegic spasticity. But, in addition to the history of the case, we have here to guide us the facts that hemiplegic spasticity is associated with paralytic symptoms affecting more or less severely the whole of one side of the body, and usually involving the face; while in lateral sclerosis a careful examination will almost invariably detect some weakness of the muscles, and increased reflexes on the other side as well. This, taken along with the distribution of the more pronounced symptoms, should protect the physician from error in this respect.

5. *From complex spinal diseases.*—The occurrence of spasticity may occasionally lead a practitioner to overlook coexisting lesions of the cord; but in every case we should keep in view the possibility of such an association, particularly of muscular atrophy, as in Charcot's disease, and of locomotor ataxia, as in the well-known combined lesion of the lateral and posterior columns.

Prognosis.—The prognosis is unfortunately always unfavourable; not that the disease of itself is a frequent cause of death, still less that it implies immediate danger, but it advances insidiously, and seldom, I believe, if ever, is really recovered from. In the early stages of the disease an arrest may take place. But in the later stages, when marked rigidity has set in, there is no room for hope of material improvement.

Treatment.—The treatment of spastic paraplegia has hitherto been eminently unsatisfactory. The most important measures are rest, careful dieting and improvement of the general tone of the system. It is surprising to observe what improvement takes place in the condition of a hospital patient who for a long time has been striving to get through the labour proper to his employment, when he gets rest in bed for a month. The relief of discomfort and the alleviation of spasticity are alike remarkable. A good and generous diet also appears to me to be of service, and such medicines as cod-liver oil, compound emulsion of petroleum with hypophosphites, iron and arsenic may improve the general health, although they may not influence the essential pathological conditions. Regulated exercises should not be neglected after the period of rest, but should never be so employed as to cause fatigue. The use of warm baths at a temperature of 90° to 95° F. affords much comfort to the patient, and often temporarily relieves spasticity. In early stages warm douches to the spine and to the affected limbs give a measure of relief. I have also seen unmistakable diminution of spasm follow the application of ice-bags over the spine, but the benefit was both small and temporary. In cases of syphilitic origin the use of large doses of iodide of potassium, alone or in combination with mercury, by the mouth or by inunction, has been followed by good results; but the great majority of cases are not of such origin, and even those which are sooner or later reach a stage at which drugs are no longer effective, for drugs cannot influence a post-syphilitic cicatrix. I think it well, however, to give every patient the chance of improvement under this treatment; not those only who confess to syphilis, but also those who, whilst denying syphilis, admit the risk of exposure.

Before going on to describe the other forms indicated in our classification, it may be well to refer to a variety of primary lateral sclerosis which was described by Professor Erb in 1895 under the name of Hereditary Spastic Spinal Paralysis. He cites as examples the cases of two sisters aged twelve and six respectively. They presented the ordinary features of lateral sclerosis as we see it in the adult, both the patients having been apparently quite healthy until the age of four years; and, although the patients are still alive, there can be little doubt of the nature of the lesion from which they suffer. Erb is of opinion that they indicate the existence of a hereditary or family group of lateral scleroses analogous to the groupings which occur in Friedreich's disease, in pseudo-hypertrophic paralysis, and a number of other hereditary nervous maladies.

II. PRIMARY SPASTIC PARALYSIS, COMPLICATED WITH OTHER LESIONS OF THE CORD

A. SCLEROSIS OF LATERAL COLUMNS AND OF ANTERIOR HORNS; AMYOTROPHIC LATERAL SCLEROSIS; CHARCOT'S DISEASE.—Charcot distinguished this form of disease from other spinal maladies, and marked it off from the progressive muscular atrophy of Aran and Duchenne. In some cases the spastic symptoms appear before the myotrophic; in others the anterior horns seem to be first invaded. As disease of this area of the cord is an essential part of the pathology of the disease it will be described later (*vide* p. 176).

B. ATAXIC PARAPLEGIA.—**Definition.**—A chronic disease of the spinal cord, *consisting* in a combined sclerosis of the lateral and posterior columns; *characterised* by the ordinary features of lateral sclerosis, namely, peculiar spasticity, with marked exaggeration of deep reflexes, rigidity, and a tendency to contracture, along with the ordinary features of locomotor ataxia, namely, staggering gait, especially marked when the eyes are closed or when the patient is in darkness, along with a variety of other symptoms characteristic of posterior sclerosis, except in so far as they are controlled and modified by the lateral lesion; *caused* by conditions not ascertained, *resulting* in gradual increase of the symptoms to a fatal termination.

Morbid anatomy.—Lesions are met with both in the posterior and lateral columns. So far as the posterior columns are concerned the lesions closely resemble those seen in the cord in ordinary pure tabes dorsalis. But the lumbar and dorsal portions may suffer equally, or the dorsal more than the lumbar; and the changes in the postero-external root zone are scarcely so pronounced as in typical cases of locomotor ataxia. With regard to the lateral columns the pyramidal tracts are found to be the centre and chief seat of the disease. But this process often extends beyond these into the lateral limiting layers, the mixed zones of the lateral columns, and the direct cerebellar tracts. The direct pyramidal tracts are also more or less diseased in almost all instances.

Clinical history.—The disease is insidious in its onset and slow in its progress. Either of the combined features may predominate at first. Patients often complain of a sense of great fatigue in the legs after a short walk, sometimes amounting to actual pain. But the lightning pains of tabes are scarcely ever complained of. Neither is the girdle sensation common. Crises are much rarer than in tabes. The Argyll-Robertson symptom does not as a rule appear, but nystagmus is not infrequently observed. Yet the unsteadiness in gait, especially in the dark, are very distinct, and the exaggeration of reflexes contrasts in a remarkable way with their habitual absence in pure locomotor ataxia. Ankle clonus, knee clonus, and other tokens of exaggerated sensitiveness to stimulation of the muscles are common. The diminution or loss of sensibility is less marked than in ordinary cases of tabes dorsalis, and the electrical reactions remain unchanged. In the course of months or years the disease comes to resemble more a pure lateral sclerosis. The rigidity prevails, the patient is unable to leave his bed, and thereby some of the characteristics of tabes are lost. But, on the other hand, in comparatively early stages, under rest and tonic treatment, the spasticity may be alleviated while the ataxia remains unchanged, and sometimes a case in which the spastic phenomena prevailed at first conforms in the end more nearly to a pure ataxia.

Causation.—The disease is proper to adult life, and prevails more in the male than in the female sex. It is most common in persons of nervous constitution, and may be supposed to result from an inborn want of vitality in certain parts of the nervous system. Sometimes it is associated with a syphilitic taint, but not so often as is the case with tabes. I am satisfied that it stands related to excessive muscular exertion, and with exposure to cold and wet, as well as to concussion or other injuries of the cord. Perhaps sexual excesses may in some instances be concerned in the production of the disease. Anæmia, leucocythæmia, and the cancerous cachexia have been associated with it in a considerable number of cases; but whether the spinal malady be a result, or both depend upon a common cause, is not yet clear. Its association with general paralysis of the insane is also of much interest. Probably the lesions proper to each may be dependent upon a common cause.

The **diagnosis** of this from other conditions does not, as a rule, seem difficult, judging from the presence of the ataxic symptoms on the one hand and the spastic on the other. Perhaps *certain forms of myelitis* may most closely simulate this disease; but in myelitis the beginning is more abrupt, and there is less tendency to the slow but steady increase of symptoms than we find in this malady. Cases of *cerebellar tumour* sometimes present considerable resemblance; but the head symptoms, the headache, the optic neuritis, the giddiness, the vomiting, the peculiar gait, like the reel of a drunken man, and the less definite spastic phenomena, should render the differential diagnosis easy. It may also be confounded with *disseminate sclerosis.* Well-marked cases of each cannot, of course, be confounded; it must be remembered that just as multiple sclerosis has been found after death in cases which showed the clinical features of spastic paraplegia, it may also present in the earlier stages symptoms akin to those of ataxic paraplegia. It seems unnecessary to point out the distinction between Friedreich's hereditary ataxia and this condition, for it should be as easy to distinguish it as it is to distinguish ordinary tabes dorsalis.

Prognosis.—The disease is not in the end recovered from; but its progress is slow, extending generally over a number of years. Death results usually from complications, such as inflammations, urinary troubles, or other intercurrent maladies.

Treatment.—Treatment is of little avail, but should be that generally employed in other scleroses.

C. PELLAGRA is a form of nervous disease scarcely ever seen in this country. Its essential anatomical changes are combined degenerations of the posterior and lateral columns of the cord, frequently associated with atrophy of the large cells of the anterior cornua, and in a large proportion of cases with chronic inflammatory thickening of the pia mater, and, sometimes, the formation of bony plates in the arachnoid. Its symptoms closely resemble those of ataxic paraplegia, but are attended with others due to the implication of structures other than those affected. It appears to be caused by the ingestion of a poison which occurs in diseased maize. In regard to the management of cases of the kind, preventive rules of course at once suggest themselves, but treatment of the central lesions is far from promising (*vide* vol. ii. p. 800).

D. PUTNAM AND DANA'S COMBINED SCLEROSIS OF THE LATERAL AND POSTERIOR COLUMNS.—This is a condition which, both from the description given by these authors and from the observation of one case which came under my own care, I believe to be entitled to recognition as a distinct malady. It may be defined as *consisting* in a subacute sclerosis of the lateral and posterior columns of the cord, passing finally into softening at certain levels; *characterised* by symptoms corresponding to those of ordinary spastic paraplegia, but more rapidly developed. Its *causes* are not ascertained. It *results* in death much more speedily than is the case with the more common form of postero-lateral disease.

Morbid anatomy.—The anatomical lesions consist in a rather rapidly advancing sclerosis of lateral and posterior columns, with associated patches or areas of more acute inflammation in the substance of the cord.

Clinical history.—The disease is one of adult life, and begins with numbness of the extremities followed by progressive enfeeblement, and is attended by great emaciation and anæmia, frequently by obstinate diarrhœa. There is at first no paralysis of any special groups of muscles, but general weakness and, finally, paraplegia set in. Sometimes anæsthesia and ataxy appear in well-marked degrees, but more commonly there is exaggeration of the knee-jerk and even ankle clonus. The legs are much more affected than the arms. The girdle sensations and lancinating pains are rarely met with. Vision and other special senses and speech are not disturbed. Mental symptoms resembling dementia occurred in the final stage in one case. The disease generally proves fatal within two years of its commencement.

The **causation** is not certainly determined, but apparently it is not connected with syphilis or alcoholism. Lead poisoning has been suggested as a cause. It is said to be more common in women than in men, and is often associated with pernicious anæmia.

Diagnosis.—In the earlier stage of the malady one may confound it with *Peripheral neuritis.* But the resemblance is only superficial, and dependent on the numbness and pains in the limbs associated with the debility. The presence of exaggerated knee-jerks and of ankle clonus, as well as the absence of other really distinctive features of peripheral neuritis, should enable us to mark it off with little difficulty.

The **prognosis** is unfavourable.

Treatment should follow the usual lines adopted in similar spinal maladies; but little is to be expected from the remedies we at present possess.

III. SPASTIC PARALYSIS SECONDARY TO PRE-EXISTING DISEASE

A. SECONDARY TO DISEASE OF CORD, TRANSVERSE MYELITIS, TUMOUR, RESULTS OF POTT'S DISEASE OF VERTEBRÆ, ETC. (*Vide* vol. vi. p. 854.)

Definition.—A secondary disease of the pyramidal tracts of the spinal cord, *consisting* in sclerosis of these tracts; *characterised* by the ordinary features of spastic conditions, particularly by the spastic gait, increase of deep reflexes, resistance to passive movements, rigidity and contracture; *caused by* pre-existing lesions in the cord above the seat of the sclerosis, and *resulting in* many cases in permanent disability, but sometimes in gradual recovery if the causal lesion be got rid of.

Morbid anatomy.—The process consists essentially in an overgrowth of the neuroglia with a corresponding atrophy of the nerve elements of descending tracts below the level of the primary disease. Of the tracts so affected the pyramidal fibres are by far the most important.

Clinical history.—In some cases the causal lesion is unmistakable; for example, an injury to the cord by direct violence, as is often seen among miners. In others the lesion may be scarcely noticeable, as in some cases of Pott's disease of the vertebræ, attended by little deformity of the spinal column. The features which mark the secondary degeneration are those with which we are already familiar—the rigidity, the exaggeration of deep reflexes, ankle clonus, resistance to passive movements, contracture, and ultimately deformity of feet and lower limbs. The condition in many cases advances very slowly, and continues for long periods to show little sign of increase. It usually lasts to the end of life, but may become arrested and markedly diminish in cases in which the cause has been got rid of, as I have seen when caries of the vertebræ has been arrested.

The **treatment** of such cases presents no likelihood of cure unless the primary disease can be arrested in an early stage. This is only possible in the case of tumours pressing on the cord, which may sometimes be removed surgically; in caries of the vertebræ, which may sometimes be successfully treated, either surgically or by prolonged rest, with the application of extension to the spine; and in the case of gummata, which may yield to medicinal treatment. If the primary disease be got rid of, the secondary may gradually diminish, but the treatment of this latter cannot be more than symptomatic, and alleviation of discomfort is really all that can be aimed at.

B. SECONDARY TO INTRACRANIAL DISEASE.

1. *Secondary to hemiplegia in the adult.*—The well-known spastic condition which sometimes occurs at the commencement of a hemiplegia, but much more commonly comes on a few weeks later, and in many of the latter kind is universally recognised as depending upon Türck's descending degeneration of the pyramidal tracts. It will be found fully discussed in a later article.

2. *Secondary to intracranial disease in infants.*—The Infantile Cerebral Palsies, pre-natal, natal, or post-natal, according to time of origin, and paraplegic, hemiplegic, diplegic, or monoplegic, according to the parts involved, constitute an interesting and important group of spastic conditions. They are described hereafter under their respective heads.

IV. FUNCTIONAL OR HYSTERICAL SPASTIC PARALYSIS

Although this subject has already been incidentally discussed when dealing with the differential diagnosis of primary spastic paralysis from hysteria (p. 140), it may be well here to state its essential features in connected form. Like other manifestations of hysteria it is much more common in the female sex, but occasionally occurs in the male and then often in a very exaggerated form. I have met with at least four clinical varieties.

The *first variety* is unmistakable by any one accustomed to study nervous cases. The patient has a history of other hysterical manifestations, is of an age at which hysteria is common, and the spastic symptoms set in suddenly and in connection with emotional disturbance.

The *second variety* is less distinct. In many respects the features are like those of the first form, but the development is insidious and the symptoms persistent. Still, in such cases, in the long run spasticity may pass away and the limbs recover their full vigour.

A *third variety* is one alike difficult and distressing. I have not seen a necropsy of such a case, but in at least one instance I have known a patient who had, as I at first hoped, merely hysterical spastic conditions, pass in the end into what I was compelled to regard as true spastic paraplegia.

The *fourth variety* is one which I have occasionally met with in people submitted to me for examination in connection with railway injuries. The history generally is that they had been perfectly well before the accident, that the accident resulted in shock to the system, and that after the accident the power of the limbs was more or less diminished. In addition are observed spastic phenomena, and often increase of the knee-jerk; sometimes ankle clonus. The question comes to be whether the patient has a progressive lateral sclerosis, or a traumatic neurasthenia of a spinal form, or whether he is malingering. Malingering is generally easily detected by one who is accustomed to examine nervous cases. But we have often to remain in doubt as to a diagnosis between lateral sclerosis and this form of traumatic neurasthenia; even the latter constitutes a formidable disease, and therefore, if clearly traceable to an accident, undoubtedly entitles the patient to compensation.

Apart from medico-legal cases, we are sometimes called upon to decide very delicate and difficult questions. For example, I had once, with the late Dr. Struthers of Leith, to decide whether a young lady, whose marriage day was approaching, should be allowed to marry or not, considering that spastic phenomena had appeared in both legs. From our examination we concluded that it was purely hysterical, and we advised that the marriage should go on. Happily speedy recovery took place.

There are certain features of hysterical spasticity which seem to me to be of much importance in relation to diagnosis.

(*a*) Contracture, and that of a peculiar form, sometimes arises at an early stage of the disease, when in an organic case it certainly would not have appeared.

(*b*) The gait shows peculiarities like those met with in other hysterical palsies. Instead of the toe-scraping of ordinary spastic disease, the whole foot is shoved forwards in walking, and the patient, instead of endeavouring to compensate for the deficiency as one suffering from organic disease learns to do, seems to make the most of the symptom.

(*c*) The knee-jerk is often exaggerated, but it is apt to be attended by spasm of the spine as well as of the limb, the patient sometimes complaining of the tap on the patella as hurting the back.

(*d*) Ankle clonus is rare, indeed very rare in functional cases, and I have never known knee clonus to be present. The pseudo-clonus presents characteristic features which have been already described.

The prognosis in hysterical paraplegia is, of course, much more favourable than in the other varieties, and recovery may be sudden or gradual. The worst cases are those which go on to contracture; and Dr. Weir Mitchell has found that the most hopeless are those in which one limb after another is attacked, until nearly all the voluntary muscles of the limbs as well as those of the trunk may become involved. Isolated contractures of single groups of muscles as of single limbs have a more favourable prognosis.

Treatment.—Functional spastic diseases are mainly benefited by moral management, and by suggestion and isolation, with massage and feeding—the general line of treatment with which the names of Drs. Weir Mitchell and Playfair are associated,—methods which are fully explained in other parts of this work (vol. i. p. 373). When a patient is to benefit from such treatment the amelioration is generally speedily seen. The longer the symptoms persist after the treatment has been commenced, the less favourable is the prognosis.

T. Grainger Stewart.

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FRIEDREICH'S ATAXIA

(HEREDITARY ATAXIA)

In 1861 Professor Friedreich, of Heidelberg, described a disease of the spinal cord which he designated as a chronic degenerative atrophy of the posterior columns, and the chief clinical features of which were incoördination, first of the lower and afterwards of the upper limbs; ultimately involving the organs of speech, but unattended by paralysis of the sphincters, or by any sensory or trophic changes. The earliest published papers by Friedreich are in *Virchow's Archiv.* Since that time numerous cases of this kind have been described by various observers, and a considerable number of statistics have been compiled.

Definition.—A rare disease of the spinal cord.

Consisting in sclerosis with marked overgrowth of neuroglia and destruction of nerve elements in the whole of the postero-internal, much of the postero-external, also of the crossed pyramidal and direct cerebellar tracts, Clarke's vesicular columns, and usually the tract of Gowers, also in some cases apparently in atrophy of the cerebellum. *Characterised by* the occurrence of the disease in several members of the same family; beginning in childhood, mostly about seven or eight years of age, very seldom after sixteen; with occasional giddiness and nystagmus, but without the other eye symptoms or the other sensory changes so often met with in locomotor ataxia; with ataxy of legs and a peculiar gait intermediate between the purely ataxic and the cerebellar, and with less difference on closing the eyes than in ordinary tabes. It spreads gradually, and sooner or later affects the arms and sometimes the neck; it is associated with muscular weakness, sometimes with contracture and atrophy; in a number of cases there is a chorea-like restlessness with jerking movements. The deep reflexes are lost, bladder troubles are rare, and the various crises are quite exceptional. Scoliosis and characteristic deformities of the extremities are not infrequent. Intelligence is unaffected, but articulation is disturbed, becoming slow and difficult, especially in pronouncing certain syllables. *Caused by* some hereditary influence; very rarely only one individual in a family affected, but direct transmission not common. Consanguinity of parents and neuropathic family history appear to exist in a considerable proportion of cases. The sexes are affected about equally. *Resulting in* gradual advance until the patient is unable to be out of bed, and ultimately terminating fatally by intercurrent disease.

Morbid anatomy.—Apart from deformities and other results of the process, the essential change consists in an extensive and peculiar sclerosis of the spinal cord. It involves, as in tabes dorsalis, the posterior columns—the posterior nerve-roots and ganglia being, however, less markedly affected; but in addition the lateral columns are usually sclerosed. In some cases also the process affects the cells of Clarke's vesicular columns and the fibres of the direct cerebellar tracts. The process further differs from that of tabes, in that Lessauer's tracts, which in tabes are always markedly sclerosed, in this disease usually escape. Déjerine and Letulle, in a recent elaborate paper, point out that the sclerosis in Friedreich's ataxia is not (in the posterior columns at any rate) the ordinary vascular sclerosis, but is essentially a pure neuroglial sclerosis of developmental origin. It appears also that in some cases atrophy of the cerebellum occurs.

Clinical history.—The disease is essentially one of early life; the period at which the first symptoms have been recognised varying from the second to the twenty-fourth year, and the vast majority of cases beginning between the sixth and the fifteenth year. In most cases the disease attacks several members of the same family, apparently affecting both sexes equally; isolated cases, although rare, do however occasionally occur. A general neuropathic family history is frequently to be made out, insanity or other nervous disease being common among the patient's relatives. Consanguinity of parents has also been traced in a certain proportion of cases. Hereditary syphilis does not appear to play any part in the causation of the disease.

The earliest and most marked symptom is ataxy of the legs, but the disorder soon extends to the upper limbs as well. Slight unsteadiness appears on standing and walking, and this gradually increases until the gait becomes staggering, like that of a drunken man. The patient reels along on a broad base; but neither does he lift the feet so high nor stamp with the heels so characteristically as in tabes dorsalis. Closure of the eyes produces little or no difference in the gait.

When the arms become involved the patient has difficulty in executing delicate voluntary movements, such as buttoning his clothes or grasping small objects. In many cases on attempting to pick up a small article the action is, as it were, overdone—the hand swoops down in a sudden, claw-like manner, the fingers being widely spread out and extended just before the grasp is made. The muscles of the back and head are also frequently affected. Irregular swaying movements of the limbs, the trunk, and the head are common; they are not active movements, but, as in ordinary chorea, occur when the patient is at rest.

Articulation almost always becomes impaired. In definite cases it is thick and monotonous, as if the patient had an unmanageable foreign body in his mouth. Jerky movements sometimes occur in the tongue; and my friend and assistant, Dr. Purves Stewart, informs me that in one case he met with abductor paralysis of the larynx with the characteristic stridor and dyspnœa. The expression of the face is often vacant, but usually not until the disease is well advanced. Intelligence is, as a rule, unaffected. Nystagmus is a very common though not a constant symptom; it is rarely present when the eyes are in the position of rest, but becomes more distinct the more the patient turns his eyes to the side. The pupil reactions are normal, and there is no optic atrophy.

The organic reflexes are normal. The skin reflexes are usually preserved, and the so-called deep reflexes are invariably absent from the outset. In some cases it has been observed that the knee-jerks were absent in non-ataxic and apparently otherwise healthy brothers and sisters of patients suffering from this disease.

Sensory symptoms are of very rare occurrence. Only in a few cases and in the latest stages of the disease has tactile anæsthesia been described. Lightning pains and "crises," like those of tabes dorsalis, never occur. Muscular sense is usually unimpaired. Sometimes there is undue sensitiveness to pain in the soles of the feet and elsewhere. There are no trophic changes in the bones, joints, or cutaneous structures. The muscles do waste in the later stages of the disease, but rather from disuse than otherwise. The peculiar deformity of the spinal column and of the feet and hands, to be described presently, are probably due, in part at least, to muscular weakness and to the contracture which afterwards sets in.

Lateral curvature of the spine (scoliosis) is common, and is probably due, in its earlier stages, to weakness of the spinal muscles. In some cases an antero-posterior curvature (kypho-scoliosis) has also been superadded.

The peculiar deformity of the foot known as "pes cavus" is one of the most characteristic features of the malady. The foot appears stumpy and shortened from before backwards, and the arch is exaggerated. Along with this the toes are usually over-extended at the metatarso-phalangeal, and flexed at the interphalangeal joints, so that the extensor tendons of the toes, especially of the great toe, are unusually prominent. A similar deformity of the hand may sometimes be observed, and may be named the "manus cava." Here also there is the same over-extension of the metacarpo-phalangeal, with a degree of flexure of the interphalangeal joints, most marked in the thumb, and producing a tendency to the claw-like hand.

The progress of the disease is invariably in the direction of more or less rapid advance of all the symptoms; but its actual duration varies exceedingly, since it has no tendency in itself to a fatal issue, unless it be indirectly from general mal-nutrition. Death is usually due to some intercurrent affection unconnected with the primary disease.

Etiology.—The causes, so far as they are known to us, have already been referred to in describing the course of the malady. They lie in some hereditary influence as yet undiscovered, in consanguine marriages, and a neuropathic family constitution.

Diagnosis.—In the majority of cases the diagnosis presents no difficulty to an observer who is familiar with the characteristics of the disease. The family distribution of the malady, the early age of onset, the nystagmus and other symptoms suffice in most cases to mark it off from ordinary tabes dorsalis. Difficulty is more apt to occur in sporadic cases, and in cases occurring in adult life; but these are rare, and with careful attention to the signs and symptoms should be readily recognised.

(i.) *From ordinary Tabes Dorsalis or Locomotor Ataxia.*—Friedreich's ataxia resembles ordinary tabes in the existence of ataxy and in the absence of knee-jerks; but it is clearly marked off from it by the absence of certain symptoms of tabes and by the presence of certain others which never occur in this disease. On the one hand, the absence of the Argyll-Robertson pupil, of optic atrophy, of the various tabetic crises, of trophic changes in joints or skin, of affection of the sphincters or reproductive apparatus, of lightning pains, and of impairment either of the cutaneous or muscular sense, together with, on the other hand, the presence of nystagmus, of the characteristic articulation, of spinal curvature, and of the peculiar deformity of the feet and hands, usually serve readily to differentiate the two diseases. Moreover, the variety of ataxy of the legs is somewhat different in the two diseases, as has already been explained (p. 153), and the arms are earlier affected than in ordinary tabes. Again, the age of the patient and the family distribution of the disease are often of material assistance in the diagnosis.

(ii.) *From cerebellar ataxia.*—There is another kind of hereditary ataxia which somewhat resembles Friedreich's ataxy, but differs from it in being dependent upon atrophic changes in the cerebellum. This variety usually starts after the age of 20—that is, later than is the rule in ordinary Friedreich's ataxia; the characteristic deformity of the feet and hands does not occur, nor is there any scoliosis. The gait is more "cerebellar" in character; it is rather reeling than ataxic. The legs ultimately become spastic, and, most important of all, the knee-jerks are not lost.

(iii.) *From insular sclerosis.*—The presence of incoördination, the impaired articulation, and the nystagmus produce some resemblance to the symptoms of multiple sclerosis; but the articulation in Friedreich's ataxia is thick, not syllabic as in multiple sclerosis; whilst in the latter we find exaggeration of the knee-jerks, ankle clonus, optic atrophy, and characteristic action tremors; and again the characteristic pes cavus of Friedreich's ataxia never occurs in insular sclerosis.

(iv.) *From chorea.*—It is only those cases of Friedreich's ataxia which present jerking choreiform movements that are likely to cause any difficulty; and even in them the absence of knee-jerks, the nystagmus, the characteristic articulation, the foot deformity, and other symptoms mark the malady off with great clearness.

Prognosis.—The prognosis, as we have already seen, is invariably unfavourable. Death occurs, however, not from the disease itself, but from intercurrent maladies attacking the enfeebled patient.

Treatment.—In this, as in other hereditary neuropathies and myopathies, treatment avails but little; it can only be symptomatic in its aims, and carried out with the hope of cheering the patient and retarding the progress of the malady. Massage and electrisation should be tried. The patient must not on any account make much exertion, but he should be encouraged to use whatever powers he has without fatiguing himself, and to go about as long as possible; for so soon as he takes to bed his enfeeblement will steadily increase, and the progress of his malady will be accelerated.

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HEREDITARY CEREBELLAR ATAXIA

Definition.—A chronic hereditary disease of the nervous system, *consisting in* atrophy of the cerebellum without sclerosis; *characterised by* an insidious onset and a gradual advance with alterations of sensory functions in respect of giddiness, abnormalities of vision, and sometimes of hearing, but especially by motor disturbance with chorea-like movements, impaired equilibration, and very marked unsteadiness of gait, commonly attended by interference with the movements of the arms and of articulation and swallowing, also with exaggeration of deep reflexes and ultimately spastic rigidity of muscles, the whole process unattended by trophic changes; *caused by* hereditary influence; *resulting in* death by advance of the disease or intercurrent maladies.

Morbid anatomy.—The cerebellum presents the chief lesion. It is diminished in volume, but not sclerosed, the change consisting in a simple atrophy of the nerve elements. It thus contrasts markedly with Friedreich's disease, in which, with or without cerebellar lesions, extensive degenerative lesions of the posterior columns, the crossed pyramidal tracts, the direct cerebellar tracts, and the column of Clarke are found.

Clinical history.—The onset of the malady usually falls later than that of Friedreich's disease. As Friedreich's is most common before, this affection usually occurs after puberty. It is true that this rule is not absolute in regard to either, but I think that it is nearly so. Twenty, thirty, or more may be taken as the common age for the commencement of this form of ataxia.

The process sets in slowly, and it makes tardy progress. When fairly established the symptoms are very distinctive. Sensory changes, although the less important, are not awanting; sometimes there may be pains in the limbs or other abnormal sensations; in some cases there is giddiness. The muscular sense is less markedly affected than in some other diseases. Romberg's symptom is often absent, though not invariably. Sight is often diminished, and muscular paralyses and nystagmus constitute marked features, especially when the eyes are turned to the side. The pupils, usually equal, sometimes show the Argyll-Robertson symptom, but in many cases the power of accommodation is also lost. Marginal scotoma is frequent, sometimes very pronounced. Achromotopsia, especially for green, has been noticed.

The acuity of vision is diminished. The fundus is often pale, with diminution of the papillary vessel and atrophy of retina and choroid. Hearing has been found deficient.

The motor functions are, however, those which chiefly deserve attention.

The organic reflexes are usually little changed, but a tendency to choke in swallowing has been noticed. The superficial reflexes vary, but the deep reflexes are always exaggerated. Voluntary motion is much affected. The patient cannot stand steadily, for he has a constant tendency to chorea-like movements. His gait is hesitating and stumbling, and while the legs are chiefly affected the arms and hands do not escape. Articulation is interfered with in consequence of uncontrollable associated muscular action. The condition gradually becomes more pronounced, and after a period of years a fatal result ensues.

Spasticity of the limbs becomes pronounced as the disease advances, but there is little tendency to scoliosis or to the deformities of feet and hands so often met with in Friedreich's disease.

Etiology.—The great point is the hereditary transmission of the disease. Dr. Sanger Brown, of Chicago, traced the malady through at least four generations, and found that it usually attacked several members of each generation. He found no preference of one sex rather than the other, but that it descended through females four times as often as it did through males. Atavism he found to be rare. He thought that in some instances a fall or injury had determined the onset.

Diagnosis.—(i.) From Friedreich's ataxia. The points which distinguish this disease from the more common Friedreich's ataxia are that it usually sets in later in life (above the age of 20), that the gait is more reeling or cerebellar in character, that the knee-jerks are not lost, that the legs ultimately become spastic, but do not fall into the characteristic deformities; neither is there any scoliosis. Anatomically it differs also in respect of the lesions in the central nervous system.

(ii.) From cerebellar tumour. The diagnosis must turn chiefly upon the family history of the patient, and to some extent upon the mode of onset of the disease; such symptoms as have been described above as occurring in a member of a family previously affected, and coming on in an extremely gradual way, is almost certainly to be referred to this category. The absence of the signs proper to intracranial tumour, the peculiar exaggerations of the deep reflexes and the condition of the pupils, the speech, and the tumultuousness of the movements are also highly characteristic.

Prognosis.—From the nature of the disease the prognosis is hopeless.

Treatment can only be palliative, and directed towards the relief of symptoms and the maintenance of the general health.

T. Grainger Stewart.

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T. G. S.

SYRINGOMYELIA

Synonym.—*Gliosis spinalis.*

History.—Syringomyelia (σῦριγξ, tube, hollow; μυελός, marrow) is a disease of the spinal cord characterised by the production of a cavity within the cord of varying length. The name was given by Ollivier in 1824; but the condition was first described by Etienne in 1546, and is mentioned by numerous writers on anatomy from that time onward. Portal (1804) was probably the first to ascribe a form of spinal paralysis to this lesion, on the basis of four cases which he observed. From his time, however, until 1860 the condition, though occasionally noted by pathologists, excited no interest. Then with the beginning of pathological study of the nervous system various hypotheses were proposed to explain the existence of cavities within the cord, and Lockhart Clarke, Vulpian, Hallopeau, Charcot and Joffroy, Leyden, Schultze (9), and Kahler (10) made important contributions to the subject. In the monographs of Roth, Wichmann, and Anna Baumler (1889), over 100 cases with autopsies were collected and analysed. The study of these cases from a pathological standpoint was soon followed by their analysis from the clinical; and in 1887 Schultze (14) and Kahler (15) established the possibility of diagnosing this condition during life. Their statements have been confirmed by clinical observers all over the world. In a monograph by Schlesinger (16) 526 references to published cases or discussions of the subject are given; and Dimitroff has added to this literature in a recent article in the *Archiv für Psychiatrie.*

Symptoms.—The diagnosis of syringomyelia rests upon the presence of three characteristic symptoms which, in the majority of cases, are present together. The existence of one of these symptoms alone should excite suspicion of the possibility of the disease being present; the presence of any two of them make the diagnosis very probable. These symptoms are—(i.) a loss of the sensations of pain and temperature in any part of the body, tactile sense being preserved in the analgesic area; (ii.) trophic disturbances in the skin, muscles, bones, or joints; (iii.) progressive muscular atrophy attended by paralysis.

In addition to these symptoms may appear (*a*) a spastic paraplegia, or (*b*) disturbance of tactile sense with pain, or (*c*) the general symptoms of transverse myelitis in case the disease invade respectively the (α) lateral, or (β) posterior columns of the cord, or (γ) its entire area. Such an extension is not uncommon, and hence these symptoms must be considered as a frequent complication.

The distribution of the characteristic symptoms of the disease will depend entirely upon the extent of the lesion in the cord. As this lesion usually begins in the cervical segments the symptoms almost always appear in the hands. If the lesion be limited to one or two segments of the cord the symptoms will be very limited; but if it extend throughout the entire length of the cord, and upward through the medulla and pons to the crus, the symptoms will be widespread and will involve the cranial nerves. The course of the disease is a very chronic one, the symptoms coming on slowly at any age, and often reaching a certain point and remaining stationary for years—the life of the patient being ended, as a rule, by some intercurrent disease; though occasionally sudden death is caused by the rupture of the cavity.

(i.) The disturbance of sensation, called by Charcot *dissociated anæsthesia,* is the chief characteristic of the disease. It is a symptom which is frequently unknown to the patient until it is demonstrated by the physician; although occasionally among the working classes, who are more exposed to injuries, the patient may have noticed that such injuries, especially burns, were not attended by pain. It is found upon examination of these persons that pricking, or cutting, or burning, or freezing of the affected area is not attended by sensations of pain, or of heat or cold; though the sense of touch is preserved. The sense of heat may be impaired when that of cold remains, or conversely. The sense of pain is a great protection to the body, giving warning of injury, and assuring care and rest of the part; hence its absence exposes these patients to the risks of serious affections of the skin and joints, the consequences of neglect of small pathological processes at their [illegible]. The sense of touch is not often affected at all; although in cases where the cavity progresses to a considerable size, and invades the posterior columns of the cord, it may become somewhat blunted. The muscular sense appears to be preserved, excepting in this last class of cases. From these phenomena of dissociated anæsthesia the conclusion has been reached that the paths of sensations of pain and temperature differ in their position from those of tactile sense, and that they pass into the central portion of the gray matter of the spinal cord soon after their entrance. It is certain

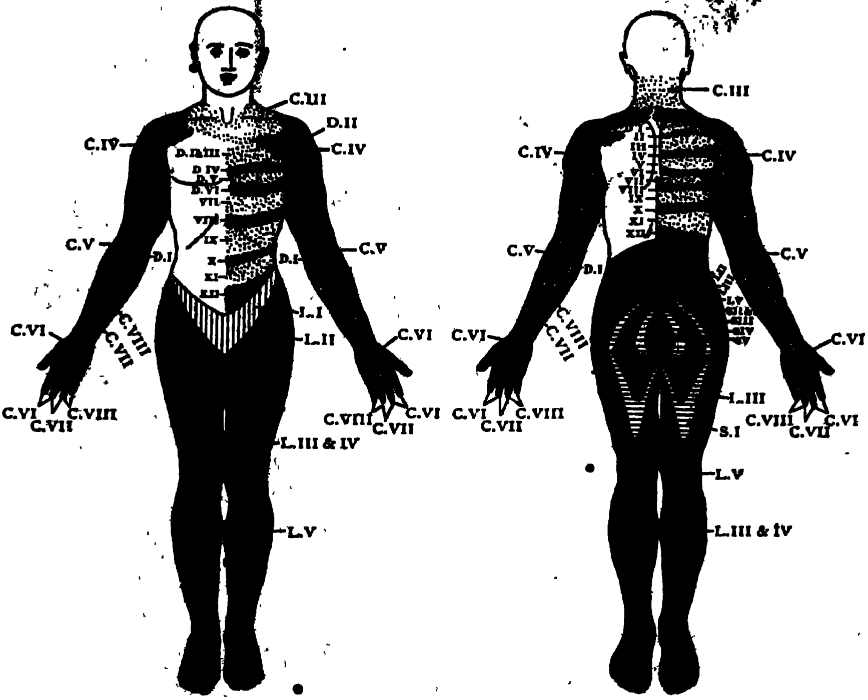

FIG. 8.—The areas of the skin related to the segments of the spinal cord.

that their entire course from below upward is not in the gray matter, otherwise a limited lesion of this portion in the cervical segments would produce a disturbance of these senses in the entire body below the lesion; but it appears that these sensations, on their way from the surface of the body to the centripetal white columns of the cord (the antero-lateral ascending tract of Gowers (?)), traverse the gray matter at the level at which they enter; hence the distribution of this disturbance of sensibility corresponds exactly to the position of the lesion in the spinal cord. As the exact area of the skin related to the individual segments of the cord has been determined (11, 29, 30), it is possible to reach a diagnosis of the precise extent of the spinal lesion by determining that of the analgesia (15). The accompanying diagram is intended to show this relation, so far as it is at present determined (Fig. 8). It will be seen that various districts of the skin can be assigned to various segments of the cord. The distribution of the analgesia in syringomyelia is usually irregular, rarely symmetrical on the two sides. Inasmuch as the affection is more common in the cervical region the condition of the analgesia is more frequently found in the hands and arms; and as it is frequently found that small injuries to the fingers are not attended by pain, this abnormality is the first to direct the patient's attention to the existence of his disease. In the early stages there is merely a decided blunting of the sensations of pain, and an inability to distinguish between slight variations of temperature; or certain sensations only are not perceived. Thus Déjerine records a case in which the thermal sense was lost for all temperatures above 68° F. The area of analgesia may not coincide exactly with that of loss of temperature sense. Paræsthesia of temperature or sharp pains rarely precede the loss of sensations. When the disease is fully established the patient cannot distinguish between ice cold and boiling water, and deep incision may be made into the flesh without the slightest pain.

(ii.) *Trophic disturbances* are a very frequent symptom in syringomyelia. In the majority of the cases it is evident that the origin of these disturbances is some injury, wound, or burn which on account of the loss of pain sense had not been observed, which had therefore been neglected, had become infected, and had gone on to ulceration or suppuration. In some cases, however, it is impossible to ascribe trophic disturbances to such causes, and indeed the hypothesis of the existence of trophic centres in the spinal cord presiding over the general nutrition and the repair of the body receives its chief support from the facts observed in this disease. The skin is the seat of the chief trophic disturbances, which may be of various kinds. There may be localised hyperæmia or anæmia of the skin; there may be changes in the perspiration, the part being abnormally covered with sweat or abnormally dry; and, in addition to the acute inflammations of the skin already attributed to injuries, cases of serous exudation with desquamation, gangrene of the skin and subcutaneous tissue, bullæ and peculiar hypertrophies and atrophies of the skin have been observed. Another trophic disturbance, which has excited much interest, is the appearance of painless whitlows upon the fingers and of small abscesses about the extremities. Morvan described a disease occurring in a seaport among fishermen, in which felons appeared upon the fingers, producing deep ulcerations and even necrosis of the terminal phalanges, and were associated with other trophic disturbances of the skin and nails, and with analgesia. *This disease, which was at first named after Morvan, is now thought to be a variety of syringomyelia;* for in all the cases examined after death a cavity has been found in the cord. The growth of the nails is commonly affected. They are hypertrophied, ridged, and occasionally stained. They become particularly brittle, and irregular in their form.

Affections of the joints and bones are very frequently observed in syringomyelia. In fact, there is no nervous disease in which joint affections occur so commonly as complications. The shoulder, elbow, and wrist are the joints most commonly affected; in this respect the disease offers a contrast to tabes, in which the joint affections most frequently occur in the lower extremities. The character of the joint affections is, however, as a rule, quite similar to that described by Charcot as characteristic of the joint disease in locomotor ataxia—a large effusion within the joint with great thickening of all the tissues, and later an absorption of the bones with an atrophy of the joint surfaces. Schlesinger has collected sixty-three cases of joint affection occurring in the course of the disease, and he estimates that this complication occurs in more than 10 per cent of the cases. Alterations in the long bones are observed in syringomyelia; and spontaneous fractures, due to a spongy and brittle condition of the bones, have been recorded by a number of observers. The joint affections and these fractures proceed alike without pain to the patient, and hence are often neglected for some time after they begin.

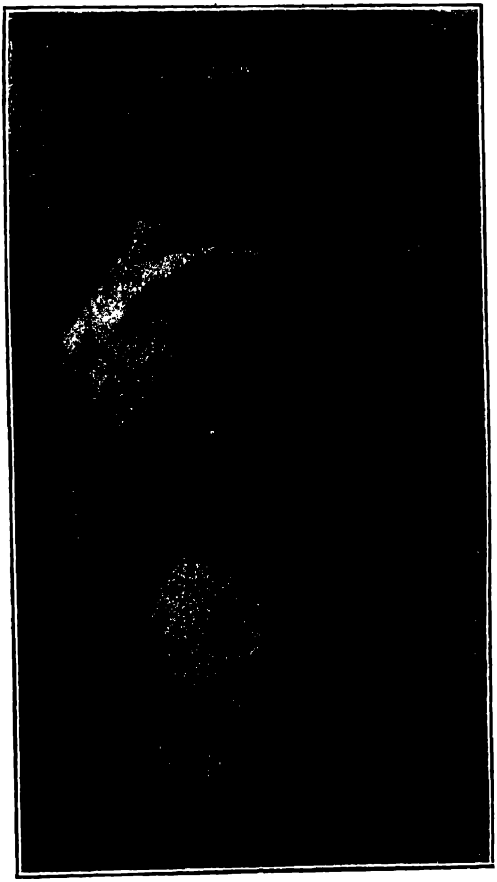

Fig. 9.—Atrophy of the arms and muscles of the back and thorax, with lordosis, in a case of syringomyelia.

In a considerable number of cases a marked curvature of the spine, either lateral or forward, and occasionally backward, has been observed. This has been ascribed by some authors to atrophy and weakness of the spinal muscles, and by others to actual changes in the bones. Both conditions may occur. The spine is, as a rule, sensitive to pressure; and deformity is more likely to occur in the upper portion of the dorsal region than elsewhere. It is never very extensive. In a few cases the combination of acromegaly with the symptoms of syringomyelia has been observed, one of these cases having been seen at my clinic; but it is by no means certain that this combination was more than accidental. As the records of the disease have increased, its incidental association with various other diseases—hysteria, paralysis agitans—has been recorded. Such associations have no particular significance.

(iii.) *Muscular atrophy attended by paralysis* is present in more than half of the cases of syringomyelia. It usually begins as a progressive muscular atrophy invading the hands, especially the first interosseous muscle, then the thenar and hypothenar eminences, finally producing claw-hands (*main en griffe* of Duchenne), and then advancing up the limb to the forearm, arm, and shoulder. Occasionally the shoulder muscles are the first affected, and then the atrophy is distributed to the deltoid and scapular muscles, and later invades the biceps and supinator longus. The muscles of the spine are particularly liable to be invaded by the atrophy and paralysis, and as a consequence curvature of the spine is a very frequent symptom in this disease. The legs are less frequently affected (12 per cent of the cases), but atrophic paralysis of the thighs or of the legs below the knee, with consequent contractures, has been seen.

The exact distribution of the atrophy and paralysis depends upon the extent of the lesion in the various segments of the cord. In a table given on pages 194, 195 (in the article on "Poliomyelitis anterior") the relation between the various muscles of the body and the various segments of the cord is shown. It is therefore evident that from the muscles invaded in any instance we may infer the extent of the lesion. The atrophic paralysis of the muscles is frequently attended by fibrillary contractions and tremors, and by a gradual diminution in the mechanical and electrical contractility of the muscle. It is not until the last stage of the disease, when the muscle is extremely atrophied, that it presents the reaction of degeneration.

(iv.) *The spinal reflexes* may be disturbed in this disease. When the symptoms are located in the arms, elbow and wrist reflexes are absent, while the patella reflex is, as a rule, increased. If, however, the disease invade the lumbar region of the cord the patellar reflex may be lost on the side of the lesion.

In a few cases in which the sacral region of the cord has been diseased a loss of control of the *bladder and rectum* occurred.

(v.) The spinal centre of the cervical sympathetic nerve lies in the first dorsal segment of the cord, and, as this segment is most frequently affected, symptoms of *paralysis of the sympathetic* of one or both sides are commonly to be detected. They are a narrowing of the palpebral fissure, a retraction of the eyeball, sluggish pupillary action with imperfect dilatation, a flattening of the side of the face, and a defective secretion of sweat.

(vi.) The extension of the disease to the medulla may cause symptoms referable to the implication of *the cranial nerves*. Atrophy with fibrillary tremor in the tongue and facial muscles, ocular palsies with nystagmus, and dissociated anæsthesia of the face and head have been observed. In a few cases paralysis of the vocal cords, disturbances in the act of swallowing, difficulty of respiration, and irregular heart action indicate that the vagus centre is affected. These symptoms are most serious, as sudden death commonly ensues (26).

The course of the disease is a chronic one. It advances slowly sometimes after an injury, and the symptoms are well established, as a rule, before the disease is recognised. The patients remain for months in a stationary condition, or the paralysis slowly increases until they are disabled. The symptoms may finally extend to the entire body, though this is rare. Death occurs either from extension to the medulla, or from cystitis or bedsores, or from some intercurrent affection; in rare cases sudden death has occurred unexpectedly from a rupture of the cord allowing an escape of fluid from the cavity.

The following histories of cases of syringomyelia under my observation at the Vanderbilt clinic illustrate the usual symptoms and course of the disease:—

1. P. S., of healthy parentage, noticed in March 1896, when he was sixteen years of age, that he was becoming weak and clumsy in his hands, that he was dropping things unintentionally, and was losing strength also in his arms. These symptoms were noticed in the left arm before they were in the right. They were not attended by pain or any noticeable sensory disturbance. It was noticed that his hands and arms became gradually thinner as they became weaker, and the emaciation soon extended to his body, especially about the muscles of the chest and back and scapulæ. He was not aware of any sensory disturbance until the time of his first examination at the clinic in January 1897. He had had no trouble in his bladder or rectum; nor any symptoms in his legs, excepting a slight weariness on any exertion; but he had noticed that his back had gradually become crooked, the right side of his body appearing to bulge. It was evident from the history that all his symptoms had made such gradual progress during the past year that they had not attracted much attention until his inability compelled him to quit work.

Examination in January 1897 showed a very marked condition of atrophy, with corresponding paralysis in the muscles of both upper extremities, chest, scapulæ, and thorax, as shown in the picture (Fig. 9). No muscle was entirely paralysed, but all the muscles were extremely weak; they presented fibrillary contractions on exposure to cold or on percussion, but did not show any reaction of degeneration. The atrophy was extreme about the muscles of the scapulæ and in the deltoids and upper part of the arms. The muscles of the thorax and back were markedly atrophied, so that a lordosis was very evident, causing peculiar motions of balancing in the act of walking. The atrophy was about equal on both sides. The biceps was less atrophic than the other muscles of the upper arm; the flexors and extensors of wrist and fingers, and thenar and hyperthenar muscles, and interossei of the hand were very decidedly atrophic. The lower portion of the pectoralis major on both sides was preserved, but the upper part was atrophic. The interossei and the muscles of the back were very atrophic. The muscles of the abdomen and legs were not in any way affected, but the knee-jerks were very much exaggerated, and there was ankle clonus on both sides. The elbow and wrist reflexes were lost. Face was normal. Sensation to touch was preserved in all parts of the extremities, body, and thorax; but sensation of heat and cold, and of pain, could not be elicited over the regions shown in the diagram (Fig. 10). The loss of pain sense was somewhat less extensive than the loss of sensation to heat and cold on the back. This patient was observed very carefully in St. Luke's Hospital for six months, there being little or no change in this condition.

2. A feeble, poorly-nourished woman, aged twenty-eight, of good family history, and without syphilis or previous illness, excepting an attack of cerebro-spinal meningitis at the age of six, first noticed the beginning of her present symptoms in 1879. Then she suffered from numbness and cold in her right hand, and soon found the hand growing weak and thin, and also quite insensitive to pain. These symptoms gradually extended up the arm and invaded the shoulder, and the muscles of the thorax and scapula; the skin of these parts becoming insensitive as the muscles atrophied. As the muscles became weak about the thorax, lateral deviation of the spinal column set in, the convexity being to the right. The hand assumes and retains the position of *main en griffe*, although the rigidity, which she says was present during the first two years, has now entirely passed away. The entire upper extremity has been wholly useless for four years. She has always noticed a trembling of the muscles. Within three months of the onset she had a bad burn on the arm, which was three months in healing, but gave her no pain. In July 1885 she had a peculiar attack:—while standing quietly she suddenly felt a numbness and tingling in the right arm, beginning at the elbow and extending up to the shoulder, neck, and face, with a feeling as though her face were drawn to the right side; this was attended by headache and vertigo, so that she felt faint, and had to sit down for a few minutes, and afterward felt quite weak for some days. She has suffered from no other symptoms, and had no return of the attack.

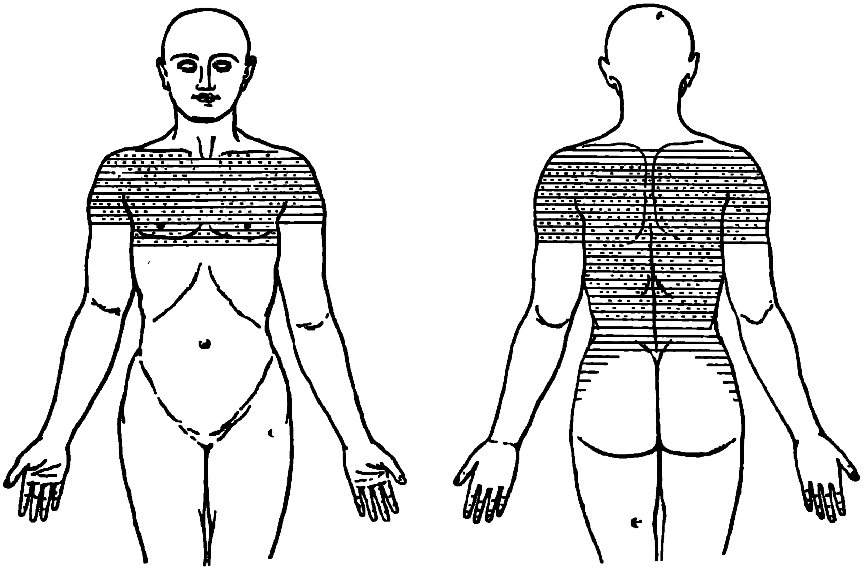

FIG. 10.—Area of analgesia and thermo-anæsthesia in a case of syringomyelia. Lines show area of thermo-anæsthesia; dots show area of analgesia.

Condition, September 1885.—The eyesight is good, the optic discs are normal, and there is no insufficiency of the ocular muscles; but the right pupil is smaller than the left, and the right eye is less widely opened, the upper lid falling slightly, and the eyeball being apparently slightly retracted. There is no facial or lingual paresis, but in the right half of the tongue there is a marked tremor. The sensation on the right side of the face, as regards temperature and pain, is less acute than on the left side, and cotton feels differently, as she expresses it, "rougher" than on the left side. Pulse and respiration are regular and normal, and she has never noticed polyuria. The right upper extremity, including the muscles of the thorax, scapula, and back, is completely paralysed and very much atrophied, but does not exhibit the reaction of degeneration. The deltoid and palmar muscles are chiefly affected, and their faradic excitability is diminished. Galvanic excitability is also diminished; for example, in the deltoid KCC = 4ma., AnCC = 7ma. The trapezius is affected, and the supraspinatus also, the latter being totally atrophied. The scaleni and splenii are not involved. The wall of the thorax is markedly thinned. The lateral deviation of the spine with elevation of the right shoulder is not due to caries, as it is not fixed, and there are no painful points; it is the result of muscular weakness in the spinal muscles. The arm hangs motionless, and all the muscles are relaxed; the only movement possible is a very slight flexion and extension at the elbow; the hand is in the claw position. Constant fibrillary tremor in the atrophied muscles is evident. No reflexes can be obtained in the arm. Sensation is very much impaired in the entire arm and upper half of the thorax. Temperature and pain sensations are suspended, but contact is perceived. Tactile sense, however, is somewhat less than on the other side; but muscular sense is not at all impaired. She feels pressure, and knows at once the position in which the fingers or forearm are placed. She can appreciate very slight differences of weight very accurately. The entire extremity is cold to the touch and cyanotic, the skin is mottled, and various scars give evidence of previous ulcerations and burns, which do not appear to have given her much annoyance, probably because they were painless. The fingers do not appear to be smaller than those of the other hand, but the growth of the nails is impaired, and they are very brittle. She does not perspire on the right side. The left arm is not paralysed, but is insensitive to heat. The right arm and forearm are each 1½ inches less in circumference than the left one. The right breast is less full, fat, and firm than the left one. The right leg does not appear to be at all weak or atrophied, but the patellar tendon reflex is very much exaggerated, and ankle clonus is easily obtained. The sensation in the right leg differs slightly from that in the left leg, cold and heat being more acutely felt, and the cotton feeling, as with the face, rougher on that side. The only symptom in the left leg is a slight increase in the patellar tendon reflex. No other symptoms. Condition stationary until 1894, when she died of pneumonia. No autopsy.

Pathology.—The post-mortem appearances in a case of syringomyelia are very characteristic. The spinal meninges are normal. The contour of the cord is sometimes irregular, owing to a bulging at some places, or a retraction at other places; or it appears at places flattened and very small: sometimes it is not altered. Fluctuation may be detected by palpation. Usually a rupture occurs in the process of removal of the cord, and the fluid, a clear serum, runs out, leaving the cord partly collapsed. It is then evident that there is a long cavity within the cord, usually near the central canal, but sometimes so extensive as, in a cross section, to leave merely a thin ring or wall of cord tissue. Sections of the cord at various levels will demonstrate that this cavity extends for some distance through the cord, and that it varies in size and shape at different levels. The usual situation of the cavity is in the lower cervical and dorsal regions. In some cases it is short, not involving more than five or six segments; in others it is long, extending through the entire length of the cord and upward into the medulla and pons. Sometimes two or three separate cavities have been found at different levels. All possible variations have been observed in different cases. In some cases a tumour has been found on one side or within the wall of the cavity, as shown in the plate on the opposite page.

When the cord is hardened, cut, stained, and examined microscopically, it presents certain characteristic appearances. The cavity may be of any size or of any shape, but lies chiefly near the central canal behind the anterior commissure, or in the posterior central gray matter, or in a posterior horn, or in both horns of the cord. In some cases it invades the central gray matter and the anterior horn or horns, but it is rarely symmetrical in its invasion of the cord tissue on the two sides. In some cases the gray matter is entirely replaced by the cavity; in other cases the cavity has invaded the white columns of one or both sides. The posterior columns are more frequently invaded than the lateral or anterior. In the most extreme cases it appears as if all the cord tissue had been destroyed; the cavity is then surrounded by a thin wall forming its sac, and no trace of gray or white substance remains. The wall of the cavity is smooth, but here and there papillary projections occur upon it.

The cavity is usually surrounded by a zone of thick neuroglia tissue which stains deeply with carmine and hæmatoxylin, is unstained by the Weigert hæmatoxylin stain, is deeply stained by the Weigert neuroglia stain, and by the Golgi stains. The thickness of this neuroglial wall varies in different cases. Its structure is more dense near the cavity; but it is thinner in the adjacent parts, and fades away into the normal cord, not usually having a sharp boundary. Under a high power of the microscope it is seen to be made up of fine fibres, of nuclei, and of small and large neuroglia cells, a few of which are seen, in some cases, to be in a state of vacuolisation and progressive liquefaction, and to be breaking down. A fine filamentous network, containing spider cells and spindle-shaped cells with long processes and many nuclei, extends outward into the nerve-tissue—the appearance being that of a partial infiltration of the normal cord by gliomatous elements, the degree of which is greatest near to the wall of the cavity. Such an infiltration of the cord with cells is also found in the segments above and below the limits of the cavity, especially about the central canal. The cavity frequently occupies the place usually taken by the central canal. Sometimes it is seen to communicate with the remains of the central canal, when some epithelial elements

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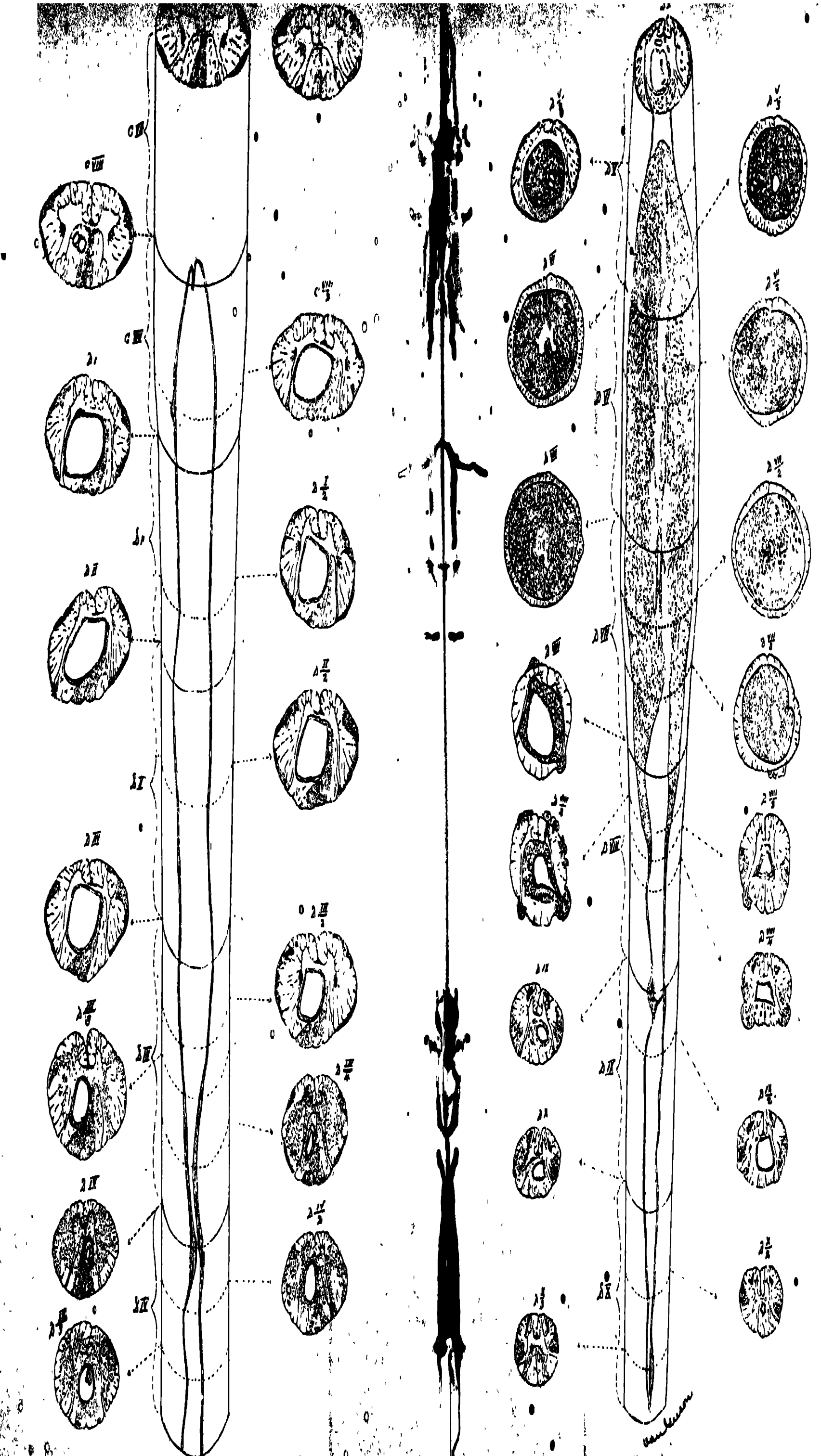

PLATE II.—The appearance of a cord in a case of syringomyelia, with glioma. (Van Gieson.)

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may be found in its wall. In other cases the canal is pushed to one side and lies in the wall of the cavity. In a few sections there may appear to be two cavities side by side; but a careful examination of sections above or below will show that one of these is really a diverticulum from the main cavity. The cavity itself is never entirely lined with cylindrical epithelium; but in a few cases one side of it was so lined, and in these there was a manifest absorption of the original central canal into the new cavity, with more or less proliferation of the lining epithelium. A few cases have been described in which a true glioma or sarcoma filled in the cavity, being an evident outgrowth from its wall (7, 31). Changes in the blood-vessels of the cord are sometimes observed. Very few capillaries are to be seen in the wall of the cavity; but outside of it, in the adjacent parts of the cord where the infiltration of small cells and nuclei is seen, the vessels are more numerous than normal, their calibre is larger, and they are more tortuous than usual. In some cases

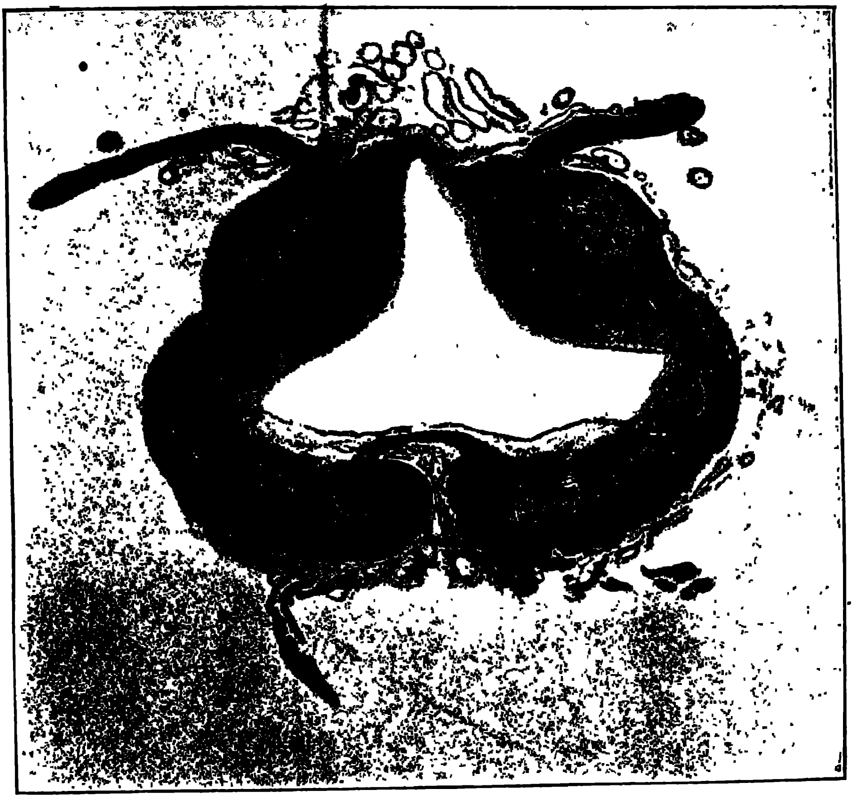

FIG. 11.—Transverse section of the spinal cord in a case of syringomyelia; first dorsal segment.

distinct thickening of their walls has been noticed. Capillary hæmorrhages are often found within the gliomatous structure.

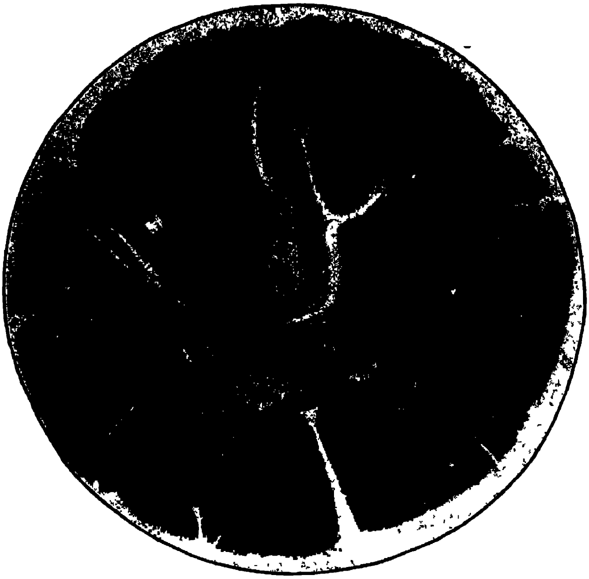

FIG. 12.—Lower thoracic region. (From *Brain*, Parts lxxiv.-v. 1896.)

Various hypotheses have been proposed to explain the conditions described. They may be briefly summarised as follows :—

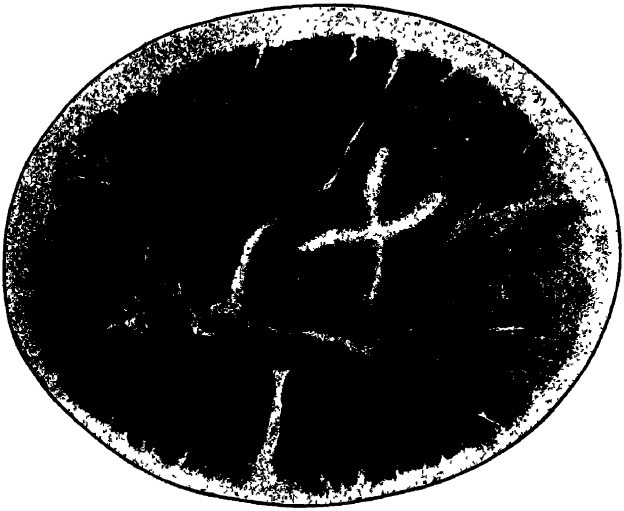

FIG. 13.—Lumbar enlargement. (From *Brain*, Parts lxxiv.-v. 1896.)

1. It has been supposed that syringomyelia always originates in a congenital defect in the development of the spinal cord. It has been

thought that during fœtal life and early infancy the central canal of the cord may be unduly distended by fluid, thus remaining as an unusually large cavity within the cord, around which cavity there is a subsequent proliferation of the embryonal epiblastic elements, or a thickening of the normal glia tissue, which is known to be more abundant in this situation than elsewhere in the cord. Such a cavity is lined with epithelium, and is recognised by many pathologists under the name of "hydromyelus." But some believe that hydromyelus may pass into syringomyelia, by the proliferation of glia cells, the invasion of the normal tissue, and its subsequent breaking down. Thus they explain the admitted fact that the degree in which the cavity in syringomyelia is lined by cylindrical epithelium is variable. Others hold that in the closure of the central canal during embryonal development a portion of it is shut off from the main canal so as to leave a subsidiary canal in the posterior septum of the cord, lined with epithelium, and surrounded, as is the normal central canal, by epiblastic tissue which subsequently proliferates, constituting a neuroglia hyperplasia, and then breaks down. Those who hold this opinion, therefore, ascribe all cases of syringomyelia to a congenital malformation of the cord, and believe that the cavity of syringomyelia has a necessary relation to the normal central canal, and usually communicates with it.

2. Another view of the disease is that the normal glia structure of the spinal cord, which is most thick about the central canal, undergoes a proliferation from some unknown cause—possibly an irritant poison in the fluid of the central canal (1); that this gliomatous new structure extends outward into the adjacent tissue, both into the gray and white matter, and subsequently breaks down in its centre, the cells becoming liquefied and disintegrated; thus a cavity originates within a gliomatous mass. This cavity at its origin has no necessary relation to the central canal; but inasmuch as the gliomatous proliferation begins, as a rule, near to the canal, the cavity usually breaks into the central canal, and thus makes a communication with it. The wall of the cavity is described as consisting of hyperplastic neuroglia with larger and smaller branching neuroglia cells, and small spheroidal cells and oval cells lying in a network of fibres, at places closely packed together, at places loosely arranged; so that it has a porous meshwork structure. This forms a sort of limiting membrane for the cavity, but beyond it within the nervous tissue there is an infiltration of glia cells. These cells are seen to be in a state of liquefactive degeneration both in the wall of the cavity and elsewhere. In some cells the nucleus is attached to or surrounded by the homogeneous remains of the cell body. Some cells are converted into sacks of fluid. Thus there is a manifest tendency in the glia cells to break down, and the cavity is the result of such disintegration. Those who hold this opinion have named the disease spinal gliosis, believing the glia proliferation to be the essential factor in the pathology. Some of these writers consider this an inflammatory process (18); others (2) deny anything more than a simple hyperplasia.

Investigations of Weigert (33) upon the structure of neuroglia seem to establish that new formations of glia may be either cellular or fibrous in structure. If cellular, the new formation is a true glioma, such as is found in tumours of the brain or spinal cord; and in such a glioma fibres are few. Weigert calls attention to the fact that this is not the structure of the glia tissue about the cavity of syringomyelia, but that the neuroglia found around this cavity consists almost exclusively of glia fibres with few cells; and that these fibres, though extending in all directions, are chiefly vertical in their course. Miura also has shown the sharp contrast between ordinary glioma, even glioma containing a cavity, and the gliomatous condition of the cord in syringomyelia. Weigert holds that the neuroglia is merely a substance produced by nature to take the place of the nerve-tissue which has been destroyed, and that its proliferation is always a sign that the nerve-tissue has primarily disintegrated. Such destruction of nerve-tissue would therefore, according to his view, precede the formation of gliomatous tissue; hence he wholly discards the hypothesis of syringomyelia to which the name spinal gliosis has been applied. Weigert says, "Many authors believe that the essential lesion in syringomyelia is the formation of a tumour followed by softening and the formation of a cavity. It is admitted that there is a growth of neuroglia of the typical fibre-type about the cavity. But this fibre mass, devoid of cells, does not resemble a glioma, and there is no reason to believe from the mere presence of neuroglia that the cavity is not a congenital or acquired abnormality of the central canal. There is a thick cluster of neuroglia fibres normally about the canal. By the pressure in this enlarged canal the nervous tissue may be destroyed, and hence a growth of neuroglia fostered. If the pressure increases the neuroglia may also be destroyed, and in its place about the cavity a hyaline formless mass may remain. The gliosis is not the essential feature, it is only a secondary result" (34). Turner (31), however, has described a case in which there is a gradual and direct transition between a true glioma and a gliomatous infiltration of the cord with the production of a cavity.

3. Many authors have observed, subsequently to disease of the spinal arteries, the formation of cavities in the cord, in their situation independent of the central canal. Thus Müller and Medin have seen a cavity in the gray matter of the cord with walls of normal nerve-tissue, with no signs of inflammatory disease or of proliferation in the glia; the size of the cavity corresponding in situation to the degree of endarteritis in the spinal vessels, and having no definite relation to the central canal. Wieting has described a cord containing numerous cavities due entirely to the low nutrition and consequent necrosis of the nerve-tissues from disease of the spinal arteries in connection with meningo-myelitis. It has been thought by Kronthal that lymphstasis within the cord, and consequent necrosis produced by transverse compression of the cord, may lead to the formation of cavities which may or may not communicate with the central canal; but this surmise is doubtful, since compression by tumours, or after Potts' disease, is not found to cause cavities. The supposition

that a cavity in the cord may be due to a diseased condition of the blood-vessels seems proven for some cases; but such cavities do not resemble those of syringomyelia, and the suggestion finds little support in Weigert's hypothesis regarding the function of neuroglia. For if this hypothesis be correct, a neuroglia growth would occur to replace the disintegrated nerve-tissue, and in the attempt of Nature to fill up the empty space would be thickest about the cavity. Necrotic cavities, however, rarely have a well-marked wall.

4. Van Gieson has recently described a condition, which he calls hemato-myelo-porus, of perforating hæmorrhage in the cord, with the production of a long, narrow cavity. Such a cavity is occasionally surrounded by thickened glia tissue. Van Gieson shows that some cases which have been described as syringomyelia have really been old cases of hæmorrhage. Turner and Mackintosh point out that the presence of a fibrin-like material, which they describe in some of the cavities in several cases of gliomatosis of the cord, suggests that hæmorrhage assists in the formation of these spaces.

It seems evident, therefore, from a review of these various hypotheses, that cavities may be formed within the spinal cord under varying circumstances, and by various pathological processes. First, from congenital defects of development; secondly, by a disintegration subsequent to a neuroglia proliferation, either of inflammatory origin or of spontaneous occurrence; thirdly, as the result of retrograde metamorphosis of tissue, the nutrition of which is impaired by obstruction to the circulation; fourthly, by actual destruction of the cord by hæmorrhage.

Lastly, there is not wanting a hypothesis which would trace it to bacteriological infection. Prus, from careful study of Morvan's disease, reaches the conclusion, which several authors had already announced, that Morvan's disease and syringomyelia are identical. He calls attention, however, to the fact that Zambaco, after a careful study of Morvan's disease, declared it to be identical with lepra anæsthetica, the bacillus of which can be recognised. And he appears to be willing to admit that the three diseases are, in fact, due to the same cause, namely, an infection of the nervous system by a germ which, in the lighter forms, attacks the peripheral nerves only, and in the more severe forms attacks the spinal cord. In this view the neuroglia formation is set up by the irritation of the bacillus, and the cavity is due to the disintegration of the gliomatous substance. This hypothesis is strongly combated by Babes, who has observed six cases of leprosy in which the bacilli were found in the cells of the cord, but in which there was no lesion resembling that of syringomyelia. The time has not yet come to establish conclusively any one of these views of the origin of syringomyelia. In fact, until cases are observed at the onset of the disease, as well as after a long duration, no basis for a conclusion can be established.

When the cavity has existed for some time it is not uncommon to find evidences of ascending and descending degeneration in the columns of the cord, which are secondary either to pressure, or to the destruction of tissue at its point of maximum extent. Degeneration in the motor nerves and atrophy of the muscle fibres are also parts of the lesion in this disease. The various trophic disturbances in the bones and skin also require mention.

Diagnosis.—When the three characteristic symptoms already described are present in any case there is no question regarding the diagnosis of syringomyelia. In the early stages of the disease, however, before all three symptoms appear, other spinal affections may be suspected. Thus many cases are regarded for a considerable time as cases of progressive muscular atrophy, or of chronic anterior poliomyelitis; and it is only on the appearance of the peculiar sensory disorder, or of the trophic symptoms in the skin or bones, that the first diagnosis becomes questionable. In other cases the early suspicion may be of tabes, especially if the disease be located in the lower part of the cord; for then the pains, especially the burning sensations, the disturbances in temperature and pain sense, and the paræsthesia, with trophic disturbances in the joints and loss of tendon reflex at the knee, may suggest tabes, even though ataxy be not manifest. Then it is only when atrophies of the muscles and paralysis concur that syringomyelia is suspected. The diagnosis from amyotrophic lateral sclerosis may be made from the fact that in this disease there is an increase of mechanical excitability in the paralysed muscles, an increase of reflex action, an early appearance of a spastic gait, an absence of sensory symptoms, and little tendency to trophic disorders.

A general myelitis or a disseminated myelitis may be diagnosed in cases of syringomyelia, when both motor and sensory and trophic disturbances are present; but the lack of symmetry of the sensory disorders and the peculiar preservation of tactile sense in syringomyelia should enable the observer to avoid this mistake. It is, however, to be remembered that in some cases the two diseases occur together (9).

While it is true that syringomyelia has a close analogy to tumour formation in the spinal cord, especially to glioma, it is to be remembered that tumours of the cord are usually limited in extent to two or three segments; that they produce more widespread symptoms than syringomyelia, especially in the body below the level of the lesion; that the symptoms resemble those of a transverse myelitis of rapid onset, and that in spinal tumours pain of a severe character is a constant symptom. The course of the disease, steadily progressive in tumour, may aid the diagnosis when symptoms are ambiguous. Pachymeningitis cervicalis (vol. vi. p. 533) may give rise to somewhat similar symptoms in the arms; but the severe pain in the neck, the rigidity and the fixed posture, the absence of dissociated anæsthesia, and the lack of trophic disturbances, will prevent any mistake in diagnosis.

Syringomyelia presents some of the features of bulbar palsy when the cavity invades the medulla and pons; but the cavity is rarely confined to the medulla and pons, and hence in syringomyelia the symptoms are not exclusively bulbar: thus is afforded a point of distinction between the two diseases.

Prognosis.—The prognosis as to recovery is unfavourable; but inasmuch as the disease rarely progresses beyond a certain point it cannot be considered dangerous to life.

Treatment.—No remedy is known which will arrest the pathological process. The symptoms are to be treated as they arise—the paralysis, for instance, as in anterior poliomyelitis. The trophic disturbances may often be prevented by care; and, if they occur, are to be treated by rest, by mechanical appliances, or by surgical measures. The sensory loss cannot be remedied by faradic applications. It is to be remembered that the disease often comes to a spontaneous standstill, so that remedies of a constitutional kind are not to be implicitly trusted, even though they appear to arrest it.

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M. A. S.

AMYOTROPHIC LATERAL SCLEROSIS

THIS affection was described by Charcot, in 1872, as a definite disease. He distinguished it pathologically, by the presence of sclerosis of the lateral columns of the spinal cord in addition to atrophy of the anterior horn cells; clinically, by rigidity of the limbs and increase of the deep reflexes, phenomena which precede the atrophy of the muscles. The atrophy was regarded as secondary to the lateral sclerosis, and hence these cases were called deuteropathic, in opposition to the protopathic cases of progressive muscular atrophy.

Etiology.—Women are more often affected than men. The age of liability varies from 25 to 50, but most patients are over 30.

As a rule, no cause can be given unless it be cold from exposure, and privation of food; a few cases have been attributed to injuries, such as falls on the back.

Symptoms.—According to Charcot's description the first symptom is rigidity, which begins gradually and invades the upper limbs first. This is attended by pains in the arms, which are rather acute, and sometimes by tingling. Soon afterwards, or almost from the beginning, the patient complains of some weakness of the upper limbs with a slight general emaciation of them, but without the particular atrophies of progressive muscular atrophy; yet the wasting is not sufficient to account for the loss of power. From two to nine months later the lower limbs also become rigid, but this symptom is not followed by atrophy, as in the upper limbs; this phase is called the second period. Lastly, symptoms of bulbar paralysis occur, forming the third period.

From the above classical description of Charcot there is much departure in different cases, according as the upper or the lower segment of the motor tract is the more affected; moreover, the atrophy is not invariably deuteropathic, that is secondary to sclerosis, as in some cases the atrophy appears first.

There are then two chief classes of cases, namely:—A. cases which begin with symptoms of sclerosis of the lateral columns, and which are followed by muscular atrophy either coming on gradually after some months, as in the above description, or occurring almost at the same time as the lateral sclerosis; B. cases which begin with muscular atrophy, and subsequently present symptoms of lateral sclerosis, especially in the legs.

The muscular atrophy in the above cases may begin in the hands, or in the face and tongue, but rarely in the legs.

A. In the first group of cases the patient complains of general weakness and inability to do as much as usual; and in some instances he complains of a certain amount of stiffness in the limbs. On examination it is found that the deep reflexes all over the body are increased. The knee-jerk is excessive on both sides, ankle clonus may also be obtained; and on striking the lower end of the radius, when the elbow is flexed and relaxed, the tap is followed by a smart flexion at the joint. It is in these cases that the deep reflex of the lower jaw is obtained, a condition which, I believe, I was the first to observe, in 1881, in a case of amyotrophic lateral sclerosis in a woman who had spontaneous clonus of the lower jaw. This reflex is best obtained by laying the finger transversely on the lower jaw just above the chin,—or placing it on the teeth of the lower jaw when the mouth is opened,—and then tapping downwards on the fingers with a percussion hammer, or with the finger of the other hand. Another method is to place a paper-knife on the molar teeth of one side and to tap it. If the reflex is increased a smart contraction of the masseters is produced, and in extreme cases a rhythmical clonus can be produced by keeping up pressure downwards on the lower jaw. This marked symptom shows that the sclerosis affects the pyramidal tract as high up as the level of the fibres going to the motor nucleus of the 5th cranial nerve. I have never seen clonus of the lower jaw in cases of any other class, except once in a case of hemiplegia.

There is also stiffness in the joints of the upper and lower limbs, and some general weakness in consequence; so that all movements are performed with diminished power.

Definite atrophy may not occur for some time, and then it may only affect the tongue (as in the case described on p. 178); or again the muscles may begin to waste very soon after the beginning of the illness. In the upper limbs the small muscles of the thumb are affected first, then the interossei and the muscles of the little finger. The order in which the muscles of the upper limb are involved is the same as in progressive muscular atrophy. Each muscle wastes very gradually, and the separate fibrils in each are successively attacked.

The atrophy of the muscles is attended by fibrillar contractions, which are most marked in the muscles in which the degeneration is just beginning.

The electrical reactions are mixed and similar to those obtained in progressive muscular atrophy. To the faradic current the muscles react as long as there are any muscle fibres left, but with increasing weakness; while to the constant current they give the reaction of degeneration, namely, a contraction with a slow response and one better obtained with the anode closure current than with that of the cathode.

Owing to the rigidity and contracture of the non-atrophied muscles, the positions assumed by the upper limbs are more characteristic than in progressive muscular atrophy; thus the fingers are bent in on the palm, the wrist is flexed, the forearm is pronated, and the elbow semi-flexed, while the humerus is kept stiffly close to the chest wall. In other cases, owing to atrophy of the interossei, the hands are markedly clawed.

The muscles of the neck may be attacked later, so that the chin falls forward on to the sternum; this is usually preceded by a certain amount of rigidity.

With regard to the lower limbs, the chief symptom is that of rigidity and spasm, and it is not usual for them to undergo atrophy of their muscles. The patient walks in a spastic manner, having great difficulty in lifting the feet off the ground, so that he drags them along after him, or he circumducts the leg, scraping the toes along the ground.

Furthermore, the patient is particularly liable to have early bulbar symptoms, due to disease of the motor nuclei in the medulla oblongata. The lower part of the face is especially affected, including the muscles of the mouth, the depressors of the lower lip, the elevator of the chin, and the orbicularis oris. The elevators of the angle of the mouth and upper lip are commonly involved. The tongue, the soft palate, and the vocal cords suffer, and in some cases the muscles of mastication. The upper face muscles, including the frontales and orbiculares oculorum, are not attacked. The wasting is preceded by a certain amount of rigidity of the lower jaw and by the presence of the lower jaw-jerk or jaw clonus; otherwise the symptoms of palsy are the same as those occurring in ordinary chronic bulbar paralysis, and they are described under that heading (p. 226).

The muscles of respiration, especially the intercostal and abdominal muscles, are apt to be involved, and thus the breathing may become almost entirely diaphragmatic. Moreover, the patient is liable to attacks or "crises" of suffocation or difficulty of breathing, and to cardiac troubles, such as increased rapidity and irregularity of the action of the heart, syncope, or sudden death. These symptoms are probably due to a lesion of the cells of the pneumogastric nucleus.

Sensation in these cases is never affected, although the patients complain of numbness and pain; tropho-neurotic changes do not occur.

The mental condition is characteristic. The appearance of the face is sad and melancholy, but as a rule these patients are exceedingly good-tempered in spite of their dreadful plight; they are also "emotional," and laugh and cry irrationally.

The arms go on wasting till there are no muscles left, or very few, and in this condition the rigidity has been observed to diminish, owing probably to the absence of muscular fibres which are able to contract.

The sphincters are never affected, unless it be at the very end of the disease.

As an instance of the condition in which the rigidity precedes the atrophy by a long interval, the following, which I have lately seen, is a very definite case of the disease as described by Charcot:—

The patient was a girl aged 25. There was no hereditary history, and no cause was given. The onset was gradual, two years ago, with general weakness in both legs; for one year weakness had invaded both arms; for nine months speech had been affected. The weakness is said to have been followed by stiffness. At present she has rigidity of the whole body and limbs, with increase of all the deep reflexes, including lower-jaw clonus, rectus clonus, and ankle clonus ; the voluntary movements are all executed slowly. The tongue is wasted, it presents fibrillar tremors, and it cannot be protruded beyond the lips. She cannot purse up the mouth. With the exception of the tongue and lips there is no atrophy of the muscles ; the hand muscles are small, but she can perform all movements with them, though very slowly. She speaks slowly also, with a nasal voice, and with very great difficulty and effort, and is almost unintelligible ; the soft palate does not move in phonation, but to reflex action, as tickling, it is very active and occurs very suddenly.

Swallowing is not affected. All the muscles react well to faradism and galvanism and KCC > ACC, though the lips and tongue react very weakly to both currents. At present, though she can perform all the movements of the legs, she is unable to walk owing to the rigidity.

The importance of the above case is the length of time that has elapsed between the onset of the symptoms of lateral sclerosis and of those of muscular atrophy ; for though it is not possible to determine the exact time when the atrophy of the tongue set in, it is certain that now, after two years, the tongue and the lips are the only parts which show signs of progressive muscular atrophy. The slowness of the movements and the weakness in the limbs are due, therefore, to the lateral sclerosis, and not to affection of the anterior horn cells. This is well shown in that, although the movements of the limbs are slow and she cannot co-ordinate her muscles for complex movements, there is no simple movement which she is unable to perform ; and she can execute all the movements of the small muscles of the hand. The same distinction is seen in the soft palate ; for, although it does not move in phonation, it acts fully to reflex irritation, showing that the motor cells in the medulla supplying the levator palati are not yet invaded. Hence this is a case of general lateral sclerosis which after the lapse of two years has been followed by atrophy in the muscles of the tongue and lips only.

B. The second class contains those cases which begin with atrophy either in the hand or the mouth, and show signs of lateral sclerosis subsequently.

I can best illustrate the chief symptoms of this class by describing a case which I published in *Brain* (Part xix. 1882) :—

A man aged 36, after much worry and exposure to cold, felt a numbness in both hands, followed in a month by bulbar symptoms, so that speech and swallowing became difficult. Six months later, when he was examined, the small muscles of the hands were almost entirely gone, and there was marked wasting, with fibrillar tremors of the extensors of the fingers and of the deltoids ; but he could move all the joints with the exception of flexing the fingers. There was no rigidity of the upper limbs, and the muscles were quite flaccid ; on the other hand, the lower limbs felt to him weak, and he walked stiffly. The bulbar symptoms gradually increased, and the wasting of the arms progressed as in an ordinary case of progressive muscular atrophy ; the legs become more rigid and the knee-jerks and ankle clonus increased, but without any decided wasting, until two years from the onset of his illness, when he became unable to walk from rigidity and leg weakness. He died suddenly, 2½ years after the onset, from impaction of food in the glottis.

This case differs from those in the former class in that the first symptom was the wasting of the muscles in the upper limbs, which was unattended by rigidity. Stiffness with increase of the deep reflexes, again, was an early symptom in the legs, in which the wasting was never sufficient to prevent some movement of all the joints. Rigidity in the legs was due to sclerosis occurring first in the lumbar lateral columns on account of the degeneration of their axis-cylinders owing to their distance from their trophic cortical cells; the absence of rigidity in the arms was due to the cells of the cervical anterior horns becoming atrophied before sclerosis had started in the cervical lateral columns. The latter view is less probable, for although the arms were not completely paralysed there was never any rigidity in them. At the necropsy, made by Mr. Victor Horsley, the cells of the anterior horns in the cervical region were found to be almost entirely gone, while in the lumbar region many of the cells were atrophied; the pyramidal tracts in the medulla and lateral columns were sclerosed along the whole length of the cord, but especially in the lumbar enlargement. The motor cells of the medulla were not atrophied.

Pathological anatomy.—In all cases of amyotrophic lateral sclerosis the affected muscles are wasted, and undergo the same changes as those described under progressive muscular atrophy (p. 215).

The cells of the anterior horns are atrophied, especially in the cervical region, as in progressive muscular atrophy. The substance of the anterior horns is altered by atrophy of the fine nerve-fibres and by proliferation of the interstitial tissue; the cells of the motor nuclei in the bulb are also atrophied (see Bulbar Paralysis, p. 231). The anterior roots, some of the motor nerves and the intra-muscular nerves, are also degenerated; but the difference between this disease and progressive muscular atrophy lies in the condition of the lateral columns.

The distinction of this disease from chronic anterior poliomyelitis has been contested by some authors mainly on the ground that the lateral columns are always affected in both diseases, and that cases of simple atrophy of the anterior horn cells are never met with. I am sure, however, that cases do occur in which the lateral columns are not sclerosed; and Charcot reported a case, noted by Gombault in 1872, in which the white substance, and the lateral columns in particular, were not altered. I have also had a case lately of progressive muscular atrophy, without contracture or increase of the deep reflexes during life, in which the cells of the anterior horns were atrophied, but there was no sclerosis of the lateral columns.

In amyotrophic lateral sclerosis there is a marked sclerosis of the pyramidal fibres, including the direct fibres in the antero-median columns and the crossed in the lateral columns, which consists of atrophy of the white fibres and of an increase of the neuroglia. The sclerosis affects the antero-lateral columns also in some cases (Hammond), but it is difficult to define the exact limits. The postero-median columns of Goll have also been found sclerosed (Charcot, Marie, Collins). A degeneration of the anterior ground bundle with sclerosis of the posterior longitudinal bundle has been described also by several observers.

In the medulla and pons, as might have been expected from the symptom of jaw clonus, there is sclerosis of the pyramidal fibres, but not so intense as that produced by a lesion of the cerebrum, and more healthy fibres are preserved. In the crus cerebri sclerosis was observed in the middle part of the crusta with atrophy of the central cerebral convolutions by Kahler and Pick in 1879.

In 1883 Koschewnikoff first showed alterations in the internal capsule in these cases, and also in the white matter of the motor convolutions, in the form of granule corpuscles resulting from the degeneration of the fibres. In the internal capsule they were found in the part occupied by the pyramidal tract. Charcot and Marie, in 1885, confirmed these observations, and found further that the large pyramidal cells of the cortex were also atrophied. It is only in certain cases that the cortex and internal capsule have been affected; in many cases the degeneration does not extend higher than the medulla.

In amyotrophic lateral sclerosis there are, therefore, two sets of changes; namely, atrophy of the cells of the anterior horns and of the motor nuclei of the bulb, with wasting of the muscles supplied by them, and a sclerosis of the lateral columns which may extend through the whole length of the pyramidal tract, even up to the motor cortex, where the large pyramidal cells may also be atrophied. It would seem as though the whole of the two segments of the motor paths were invaded separately. The only objection to this supposition is that in many cases the nutrient cells of the upper segment, that is the cortical cells, have not been found affected. It seems possible, therefore, that degeneration and atrophy of the pyramidal fibres can occur by themselves without appreciable alteration of their nutrient cells; a condition which at present cannot be satisfactorily explained, but which may be elucidated by the more recent methods of staining.

With regard to the cause of the degeneration of the cells reference must be made to a case published by Dr. Mott:—

The case began with numbness and coldness of the right leg, which soon wasted; then, in four months, of the right arm; later of the left leg, and last of the left arm: ten months from the onset there was complete loss of power in the legs and arms with great muscular wasting, and the same in the back and neck muscles. There was considerable rigidity of the shoulder, elbow, and wrist,—whether this came on as a primary or secondary symptom is not stated,—and the deep reflexes were increased. On post-mortem examination atrophy was found of the anterior horn cells, and especially of the anterior and internal groups; the peripheral nerves showed degeneration of a very few fibres. In the medulla the hypoglossal, the lower facial, and the spinal accessory nuclei were degenerated. Sclerosis of the direct and crossed pyramidal tracts and atrophy of the ground fibres occurred in the cord; and sclerosis of the pyramidal tract extended up to the internal capsule in which there were a number of degenerated fibres. Sections of the cortex of the central convolutions showed numbers of granulation corpuscles, also that many of the fibres had disappeared or were undergoing degeneration, while the pyramidal cells were absent. The pia mater over the central convolutions, and to a less degree over the spinal cord, was thickened and congested, and revealed a condition of chronic periarterial inflammation, the walls of the small vessels being thickened and hyaline; there were also many small hæmorrhages.

Mott considers that a simultaneous degeneration of the upper and lower segments of the motor path occurred, and that the degeneration of the cortical cells might be caused by the periarteritis interfering with their protoplasmic processes.

According to the researches of Golgi, Ramon y Cajal, and other recent observers, there are two distinct segments of the motor tract. The upper consists of the pyramidal cells in the cortex of the ascending frontal and parietal convolutions and their axis-cylinder processes, which extend through the internal capsule, crus cerebri, and pyramidal tracts, down to the level of the cells of the anterior horn, and around these cells the end of the axis-cylinder forms an arborisation without joining the cell. The lower segment consists of the cells of the anterior horn and their axis-cylinder processes, which are prolonged through the anterior roots, and end in the muscle as its motor nerve. It will thus be seen that it is possible for disease to start either in the upper or in the lower segment, or in both. The question is, which part of the segment is first involved? It seems certain that if the cell itself be seriously affected, its whole axis-cylinder process must go with it; while, on the other hand, if the nutrition of the cell be very gradually impaired, the part to suffer first would be the part farthest from the cell, the trophic centre or the source of nourishment for its processes. In this way we might, perhaps, account for the end of the axis-cylinder farthest away from the cell being most affected, and also for those cases where sclerosis of the pyramidal tract has not been traced upwards higher than the pons or crus cerebri. It was pointed out by Golgi that the protoplasmic processes of the cortical cells are connected with lymphatic spaces which are in connection with the perivascular sheaths; and Mott suggests that the periarteritis observed in his case might interfere with the nutrition of these processes, so that the cell would suffer, and degeneration might thus occur.

The degeneration of the one segment cannot, therefore, be looked upon as secondary to the other; though in both the degeneration is probably due to the same cause, the two segments appear to be independent. All we can say is that in the cases of the first class above described the degeneration begins in the upper segment—lateral columns, pyramidal tract, and cortical cells; and that the lower segment—anterior horn cells, anterior roots, motor nerves, and muscles—is attacked subsequently: in the second class of cases the lower segment is probably attacked first, and afterwards the upper. It has been said that simple atrophy is never followed by rigidity or other signs of lateral sclerosis;

but in the case which I have cited (p. 179), while there was never any rigidity of the arms, this was the first symptom in the legs, and appeared after the atrophy of the upper limbs.

Diagnosis.—This has to be made from other forms of muscular atrophy, due to lesions of different parts of the nervous system; and, beginning from the periphery and going upwards, mention must be made of myopathies, peripheral neuritis, lesions of the spinal roots, pachymeningitis, pressure on the cord from Pott's disease, chronic poliomyelitis, acute poliomyelitis, syringomyelia, transverse myelitis, disseminated sclerosis, and primary spastic paralysis.

The increase of all the deep reflexes will help to separate the disease from several of the above, and especially from the myopathies, peripheral neuritis, lesions of the spinal roots, simple progressive muscular atrophy and poliomyelitis, in which cases the deep reflexes are absent or are not increased. On the other hand, the deep reflexes are increased in the rest of the list; but of these pachymeningitis frequently occurs after an accident, the onset is more rapid, there are severe neuralgic pains with numbness down both upper limbs, and painful spasms in the limbs with rigidity of the neck, while sensation may be affected; these symptoms are followed, in two months or so, by atrophy of the small hand muscles, but there are no bulbar symptoms. In Pott's disease there is evidence of caries of the vertebræ at the level of the eighth cervical or first dorsal, causing atrophy of the small hand muscles; and although there might be increase of the knee-jerks and ankle clonus, there would be no increase of the deep reflexes in the upper limbs. In syringomyelia there are peculiar sensory changes in the limbs and trunk, tactile sensibility being preserved, while impressions of pain and temperature are lost; frequently also there are trophic changes in the joints and skin, and usually atrophy of the small hand muscles, but no rigidity or increase of the deep reflexes in the upper limbs. Transverse myelitis rarely causes atrophy of the hand muscles, while sensation and the sphincters are frequently affected; the onset is also more rapid. In disseminate sclerosis and primary spastic paralysis atrophy of the hand muscles does not generally occur; while in the former disease marked incoördination, nystagmus, and the characteristic speech will be unmistakable.

Prognosis.—The increase of the deep reflexes in any case of progressive muscular atrophy makes the prognosis considerably less favourable than if these symptoms were absent; that is, the prognosis of amyotrophic lateral sclerosis is worse than of progressive muscular atrophy, for whereas the latter may last some years, the former does not run more than one to three years.

The progress of the case is gradually from bad to worse, and after a time the patient is unable to walk, owing to the rigidity of the legs, and he becomes quite helpless. If the bulb be affected, death usually occurs from choking or paralysis of the respiratory centre, or from pneumonia or bronchitis, if the muscles of respiration are involved. In some cases these muscles fail directly from atrophy.

Treatment.—No drug has been found to stop the course of this disease; strychnine, which has been found useful in progressive muscular atrophy, would increase the spastic condition and the deep reflexes. The best treatment is to give tonics and cod-liver oil. Great attention is required to ensure that the patient do not choke whilst being fed. If necessary, nourishment must be given by a soft tube passed through the nose. Sir T. Grainger Stewart and other authors recommend the use of the constant current to the affected muscles, and massage to diminish the contractures.

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C. E. B.

SENILE PARAPLEGIA

A FEW years ago my attention was caught by a note of Sir William Gowers on a certain loss of power in the legs which is often to be seen in old people. It may be said that loss of power in the legs, as in other parts, is a feature of old age which needs no specification. But this is not quite so; since reading the note I have readily become convinced that the loss of walking power in the aged is very often, very often indeed, a true palsy, depending on some local decay of the spinal cord. Such persons presenting no overt signs of paralysis agitans, or like degeneration of a widespread kind, often lose the use of their legs in a degree out of proportion to the debility of other parts, even allowing for the weight these limbs have to bear. I have convinced myself of this interpretation of weakness of the legs in a very large number of cases; the disability seems to be one very apt to occur in the old. Man or woman, one who reaches advanced years—and the stage of old age is not the same for all—after falling for some months into a shuffling gait, rather quickly finds himself unable to walk at all, or is only able to drag himself along with difficulty. That this is not merely one aspect of the general decay of all parts in the aged is proved by its absence in many equally ancient and equally decayed elders, who, however, are not in any especial sense decrepit in the legs. It was but the other day that I noticed an old gentleman well above eighty years of age standing for a quarter of an hour beside a table interesting himself in some pieces of bric-a-brac without the least failure of the legs; yet a few days before I had noticed another old friend, quite as brisk on the whole, who, without suspecting he was palsied, informed me in conversation that he was no longer able to walk out on account of a somewhat sudden failure of his legs. He had been set down from a fly, as usual, some mile or so from home, that he might take his walk; but after walking for a few yards he found he could go no farther, and he had to sit down till the fly could be brought up. From that day the weakness had increased until he could do no more than creep about his house by hold of the furniture or the stair rail. In my experience, at any rate, many old people are able to walk fairly well until, within the space of a few days or weeks, they are reduced more or less quickly to a very feeble gait, if not to overt paraplegia. The disability is put down to old age, and regarded as a mere incident in a general failure of power; but the aged person finds himself confined to his chair, while, perhaps, the rest of his faculties may yet for some time be preserved, and suffice for a tranquil enjoyment of life.

The bladder of old people is so apt to be leaky that it is hard to say when such a condition is associated in any direct way with the paraplegia. In my opinion this paraplegia is so associated rather by accident. I have at present under my observation two old men, both well over eighty years of age, one of whom can get out of bed and walk about his room, and even in his grounds, but in whom the bladder is so weak that it is impossible even with constant watchfulness to prevent accidents; the other, who was rather suddenly taken off his legs, retains his water with comfort. The patient who still has the use of his legs is in every sense farther gone in senility, though not in years, than his paraplegic friend; yet even in this latter, had I not read Sir William Gowers' note, I should scarcely have realised that the inability is a paraplegia; this, however, it most probably is. This senile paraplegia is not attended with disproportionate wasting, sensory disturbance or alteration of reflexes. Sir William Gowers seems rather disposed to place the seat of the disease in the cortex.

EDITOR.

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ED.

II. NUCLEAR DISEASES

POLIOMYELITIS ANTERIOR ACUTA

SYN.—*Infantile spinal paralysis, Regressive paralysis, Acute atrophic paralysis, Atrophic spinal paralysis*

ANTERIOR poliomyelitis is an acute disease observed most commonly among children, but occasionally in adults, characterised by sudden complete loss of power in one or more limbs, followed by wasting of the muscles paralysed, and by interference with the growth of the parts, but not attended with any sensory disturbance.

Symptoms.—It usually begins like an acute infectious disease with fever, sometimes attended by convulsions, especially in infants; sometimes by considerable pain in the back, body, and limbs; sometimes by digestive disturbances, vomiting and diarrhœa; sometimes merely by general malaise. The temperature rises rapidly to 102° or 103°, and the patient may have a chill followed by sweating. The temperature remains about 101° or 102° for several days, with slight morning remission, then gradually sinks to normal, the entire febrile movement rarely lasting more than a week. Within a day or two of the onset paralysis sets in, usually in both legs, or in both arms, or in one limb alone, or in all four extremities. If the child be young, and confined to bed by the fever, the paralysis may not be noticed until the second or third day. In older children and adults the paralysis is usually manifest within twenty-four hours of the onset. It is generally observed that children cry a good deal during the period of onset, and some of those who are able to complain say that they suffer from pain in the back and in the affected limbs. This pain may remain for some weeks. Occasionally there is some rigidity of the spine or neck suggestive of meningitis, but this soon subsides. There is no disturbance of the bladder or rectum, though rarely retention of urine has been noticed for a few days. There is no tendency to bedsores or to trophic changes in the skin. There is no complaint of numbness or of paræsthesia, and there is never any loss of sensation; but the limbs are sometimes painful upon any movement, especially in the joints.

After the fever, with its attendant malaise and digestive disturbances, has subsided, and the general health has been restored, there remains a paralysis more or less extensive. This paralysis is usually more extensive at the onset than it is destined to be permanently. Thus the child may at first be completely helpless, and later recover power in all but one limb; or the trunk may be paralysed at the onset, but not permanently affected. Both legs are commonly affected together, but the final paralysis is found in one limb only. Occasionally the neck muscles are distinctly weak, and there may be difficulty in swallowing. This is seen in cases in which the arms are paralysed, and yet the final paralysis may affect but one arm. The face has been paralysed with the arms, and the ocular muscles also, but these are rare occurrences. In a number of cases in which the final paralysis was limited to two or three muscles the original paralysis was widespread, involving all the limbs. In giving a prognosis in the early stage this fact should be remembered. Sometimes the onset of the paralysis is not sudden, but there is a gradual increase during a week or ten days, then a stationary period, and then a regression. The subsidence of the paralysis begins from a week to two months after the onset, and then goes on steadily; but it is not until after the lapse of three months that it is possible to determine what muscles will eventually recover. There is always a certain amount of permanent paralysis.

The muscles which are paralysed undergo atrophy, which is more rapid and complete in those that are to be permanently paralysed; and the change in the size of the limbs is well marked within a month. The paralysed muscles are relaxed, never rigid; and to the electrical tests they show a reaction of degeneration. The reaction of degeneration consists n a loss of response of both muscle and nerve to faradic stimulus, and a loss of response in the nerve to galvanic stimulus. The galvanic reaction of the muscle remains, but the normal contractility of such a muscle to galvanic currents is altered. For the first few months the muscle responds too strongly to galvanism, and contracts under the positive pole more quickly than under the negative pole (vol. i. p. 538). Later the contractility to galvanism progressively decreases until, in a totally paralysed muscle, it is lost. It may be stated as a prognostic sign that the muscles in which the faradic reaction is preserved, though paralysed for a time, will recover. Such muscles also preserve their tone, so that they contract when sharply percussed with a hammer.

The circulation in the affected limb is considerably impaired, and it is cold, blue and flabby, but not œdematous. In some cases the bone is subsequently hampered in its growth, so that in after-life the limb is shorter and more slender than its fellow.

While the description just given of an acute onset with fever applies to about three-quarters of the cases of anterior poliomyelitis, there remains one quarter in which there is no febrile onset. Of 100 consecutive cases in my clinic, 69 began with fever, and 31 began without fever. Sinkler reports 178 with fever, 40 without fever. In these cases the child, while in a state of perfect health, is suddenly paralysed in one or more limbs. It gives no sign of pain, it does not appear to be ill, and the paralysis surprises the mother by its sudden onset. In these cases the paralysis is soon followed by atrophy and by vasomotor paralysis. It is not attended with pain or tenderness on motion, and usually decreases to some extent, leaving the limb, however, in part permanently paralysed.

These two modes of onset of the disease are evidently quite distinct, and their pathological basis is probably different.

I have said that after the onset is over there is a slow progressive improvement up to a certain point, when the permanent condition of paralysis is found to vary greatly in different cases. Its situation is usually in the legs, and here two types of the disease may be recognised—the leg type and the thigh type. In the leg type the peronei, alone or with the anterior tibial muscles, are most commonly affected,

FIG. 14.—Paralysis and atrophy of the right leg due to anterior poliomyelitis. The imperfect growth, six years after the onset, and the secondary talipes are evident.

although the posterior tibial group may share in the paralysis, or, indeed, may be as fully paralysed as the others. As the paralysis persists, deformities of the ankle and foot will appear; the form of subsequent talipes depending upon the muscles chiefly paralysed. In the thigh type the ilio-psoas and iliacus muscles and the glutei, and muscles about the thigh are those chiefly affected; the muscles on the inner side of the thigh and the muscles below the knee often escape. In these cases the leg hangs like a flail from the body, and cannot support the weight at

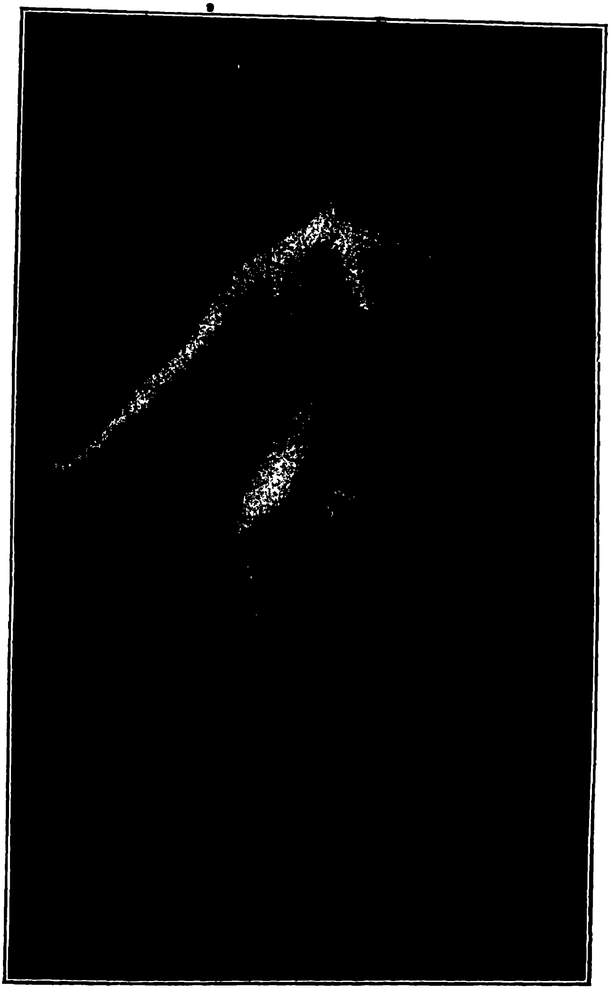

FIG. 15.—Paralysis and atrophy of the right leg due to anterior poliomyelitis. The secondary curvature of the spine due to the shortness of the paralysed limb is well shown.

all. In some cases nearly all of the muscles of the lower extremity are paralysed, and the atrophy is uniform throughout the limb. In these severe cases it is not uncommon for the muscles of the back and abdomen to share in the paralysis and atrophy. Figs. 14 and 15 show the appearance of limbs paralysed and atrophic; Fig. 14 showing a secondary talipes, and Fig. 15 showing a secondary lateral curvature of the spine due to the shortening of the leg.

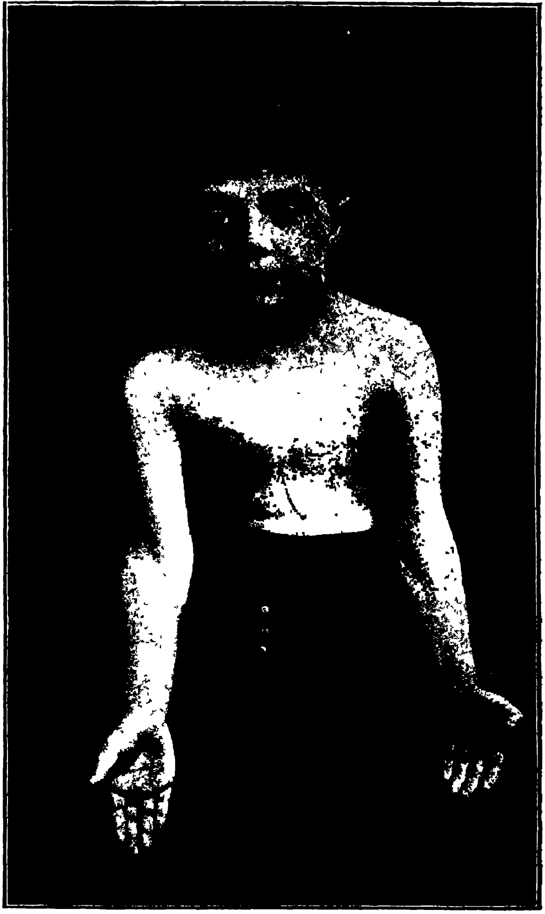

FIG. 16.—Paralysis and atrophy of the left arm and hand due to anterior poliomyelitis. The partial dislocation of the head of the humerus due to deltoid paralysis and the abnormal abduction of the thumb are evident.

When the arms are invaded two types of paralysis have been described, the upper arm type and the lower arm type. In the upper arm type the muscles about the scapula, the deltoid, the biceps, and supinator longus are paralysed and atrophic, and consequently the motions of the shoulder and elbow joints are seriously hampered. In these cases the shoulder-joint is unduly movable, and the head of the humerus falls out of the socket. In the forearm type the muscles below the elbow are invaded; the flexors or extensors of the wrist and fingers, or both together, are affected, the supinator longus escaping. In other cases the interossei muscles of the hand and the thenar and hypothenar muscles are paralysed, while the long flexors and extensors escape. Occasionally a combination of upper and forearm types occurs, in which cases the whole limb is useless. Fig. 16 shows a case of total paralysis of the arm, with atrophy and falling of the head of the humerus out of its socket in consequence of the paralysis of the deltoid. The claw-hand is also present. The upper part of the trunk is occasionally involved in the paralysis, together with the arms. Rarely the muscles of the back and trunk only are permanently paralysed.

In a very few cases the entire muscular system of the body appears to be affected by this disease; both legs, the trunk, and both arms are more or less paralysed: yet even in these cases a careful examination will show that the degree of the paralysis and atrophy is not the same in all the muscles. The relative frequency of paralysis in different parts of the body is shown in the following table:—

Table I.—Distribution of Permanent Paralysis in Anterior Poliomyelitis.

	Duchenne (1).	Seeligmüller (2).	Sinkler (3).	Starr.	Total.
Both legs	9	14	107	40	170
Right leg	25	15	63	20	123
Left leg	7	27	62	27	123
Right arm	5	9	5	7	26
Left arm	5	4	8	4	21
Both arms	2	1	1	2	6
All extremities	5	2	35	5	47
Arm and leg, same side	1	2	26	4	33
Arm and leg, opposite sides	2	1	1	4	8
Trunk	1	.	22	3	26
Three extremities	...	.	10	2	12

In addition to the paralysis and atrophy there is in every case a loss of reflex action at the level of the lesion. The skin reflexes usually return after a time; but the deep reflexes are absent for a long period, even when a partial recovery of the muscle involved has taken place. Thus the knee-jerk is uniformly absent when the thigh muscles are paralysed, and the elbow and wrist jerks when the arms are affected.

Sensation is preserved in almost every case; but I have so frequently observed a permanent hypersensitiveness to painful impressions in the paralysed limb, that I cannot but believe that the lesion in the gray matter affects the pain-sense tracts in their passage through the cord at the level of their entrance, and has a relation to this symptom. There is a marked vasomotor paralysis producing a permanent lowering of the surface temperature and a lack of vasomotor response in the limb to applications of heat and cold.

Deformities of the joints are a common sequel in infantile spinal paralysis. The approximation of articular surfaces in health is secured in part by the tonic action of the muscles, especially at the shoulder, hip, and knee; hence paralysis of the muscles controlling these joints is attended by relaxation and a greater degree of mobility than normal. Thus when the deltoid is paralysed, the head of the humerus falls from its socket, as shown in Fig. 16; and abnormal extension of the knee is often seen in the upper leg type of palsy. After some months of paralysis the muscles which are the natural opponents of the paralysed muscles are apt to become permanently contractured, and this condition also brings deformities about. In the case of the foot the action of gravitation on a flaccid part of the limb combines with the contracture to increase the deformity there; hence any of the forms of talipes may ensue on infantile paralysis (see Fig. 14). Similar deformities of the wrist are also observed, but these are not common. Curvature of the spine, from paralysis of the muscles of the back, is frequently seen, all the varieties of this change having been described (see Fig. 15). They differ from those due to bone disease in that they do not persist during suspension of the body by the head and arms. One of the most important points in treatment is to prevent the establishment of these deformities.

The progress of the disease in any case may be divided into stages. After an acute onset there is a stage of maximum intensity, lasting from one to six weeks, and followed by a period of steady improvement, which may extend from six months to a year. Then follows the permanent chronic condition in which the normal growth of the child may lead to a slow development of the limb, but not to any change in its power of use. It is very rare for a complete recovery to take place after an attack of infantile paralysis. Even in the lightest cases there is usually some weakness, slight atrophy, and coldness left; and one or two muscles will be particularly feeble. In the majority of cases considerable permanent paralysis remains, requiring the use of apparatus to assist the use of the limb, and to prevent deformities. Death has occasionally occurred during the acute onset, but is very rare; and, once this stage is passed, there is nothing in the disease to threaten life.

It is the chief characteristic of the atrophic paralysis in this disease that it selects certain muscles to the exclusion of others. This selection bears no relation to the arrangement of muscles in the limb, or to the conjoint action of muscles in producing any definite movement. It is wholly dependent upon the arrangement of the groups of cells controlling the different muscles in the anterior horns of the spinal cord.

In order, therefore, to understand the symptoms of the disease a short consideration of the motor elements of the cord is necessary. The neurons which preside over the motion and nutrition of the muscles lie in the anterior horns of the spinal cord. They are not scattered irregularly through these horns, but they are grouped together in definite clusters. A series of sections of the cord made from above downward demonstrates that the number of these groups varies in different segments of the cord, there being a large number of such groups in the cervical and lumbar enlargements, and fewer in the dorsal and upper cervical regions. Figs. 17 and 18 demonstrate this grouping of cells in the cervical and lumbar regions. Some groups of cells are very long, extending through three or four segments; while others are short, hardly reaching through one entire segment. It is evident that a lesion limited to a single segment of the cord may entirely destroy a group of cells limited to that segment, and may destroy only in part a column of cells extending into adjacent segments. If each group of cells represents a muscle it becomes evident at once that the degree of paralysis in that muscle will be determined by the extent of the lesion in its group of cells. If a group of cells is entirely destroyed, its muscle will be totally and permanently paralysed. If a group of cells is but slightly affected the muscle may be weakened and slightly shrunken, but still able to perform its work. The following table, prepared by a careful comparison of a very large number

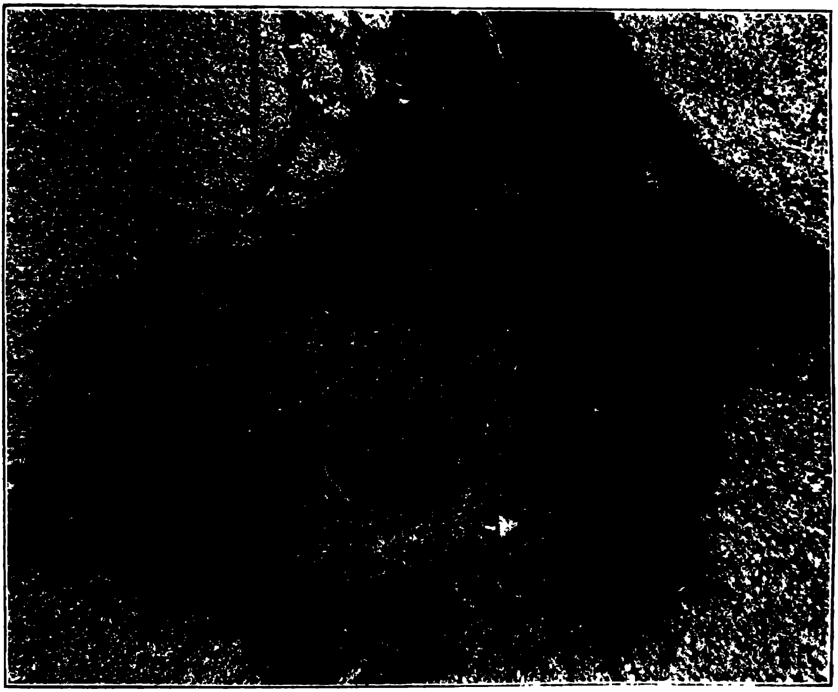

FIG. 17.—Section through the sixth cervical segment of a normal cord, showing six groups of cells in the anterior horn.

of cases with accurate autopsies, shows the situation of the various groups of cells controlling the various muscles of the body in the different segments of the spinal cord (Table II.) It will be seen that some muscles are represented in two or even three segments of the cord, while other muscles are represented in but one segment alone ; and again it will be seen

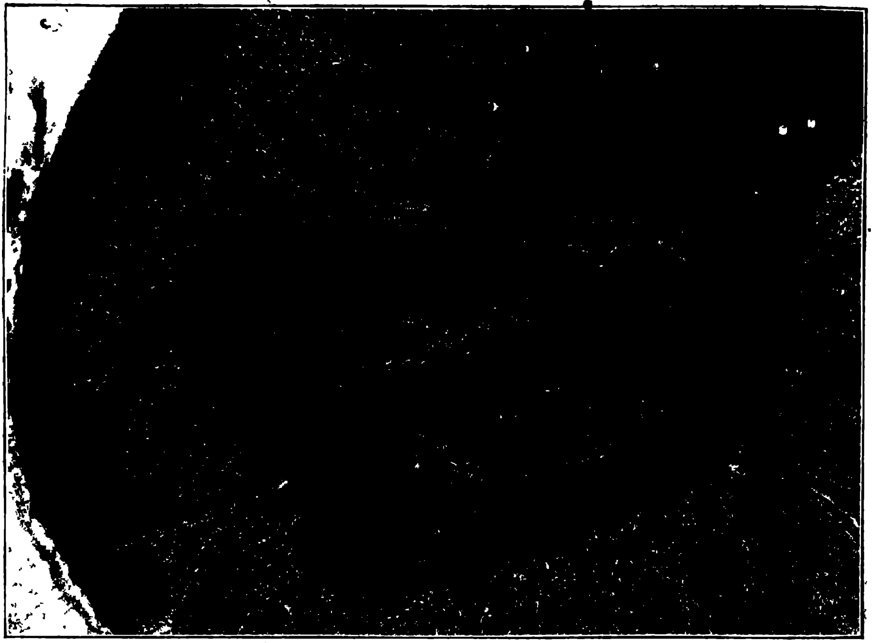

FIG. 18.—Section through the fourth lumbar segment of a normal cord, showing three large groups of cells.

TABLE II.—Muscles represented in the Segments of the Cervical Enlargement.

4 C.	5 C.	6 C.	7 C.	8 C., 1 D.
Diaphragm.	Supin. brevis.	Pronators.	Pronators.	
Lev. ang. scap.	Teres minor.	Triceps.	Triceps.	
Rhomboid.	Rhomboid.	Brach. ant.	Brach. ant.	
Supra spin.	Supra spin. }	Long extensors of wrist.	Long extensors of fingers.	Extensors of thumb.
Infra spin.	Infra spin. }			
Deltoid.	Deltoid.	...	Pector. (costal).	Intrinsic muscles of hand.
Supin. long.	Supin. long.	...	Latis. dorsi.	
Biceps.	Biceps.	Biceps.	Teres major.	
	Serratus magnus.	Serratus magnus.	Long flexors of wrist.	Long flexors of fingers.
	Pector. (clav.)	Pector. (clav.)		

Muscles represented in the Segments of the Lumbar Region.

1 L.	2 L.	3 L.	4 L.	5 L.
Quadr. lumb.	Sartorius.		Glutei.	Glutei.
Obliqui.	Quad. ext. cruris.	Quad. ext. cruris.	Tibialis ant. (?)	Biceps femoris.
Transversalis.	Obturator.	Obturator.		Semi-tendin.
Psoas.	Psoas.	Adductores.	Adductores.	Popliteus.
Iliacus.	Iliacus.			

Muscles represented in the Sacral Region.

1 S.	2 S.	3 S.	4 and 5 S.
Biceps fem.	Tibialis anticus (?).		Sphincter ani et vesicæ.
Semi-membran.	Peronei.		Perineal muscles.
Ext. long. dig.	Intrinsic muscles of foot.	Peronei.	
Gastrocnemii.	Gastroc.	Intrinsic muscles of foot.	
Tibialis post.	Tibialis post.		

that different muscles are represented side by side in the same segment. If to this table the clinical picture of infantile paralysis be referred, it will become manifest at once that the distribution of the paralysis and the situation of the lesion bear a definite relation to one another. Thus it is evident that the upper arm type of paralysis is produced by a lesion of the fifth and sixth cervical segments; that the forearm type of paralysis is produced by a lesion of the seventh and eighth cervical and first dorsal segments. It is evident again that the thigh type of paralysis is produced by a lesion of the upper lumbar segments, and that the leg type of paralysis is produced by the lesion of the lower lumbar and sacral segments.

The larger the extent of the lesion in the cord, the greater the number of muscles that will be paralysed. The more complete the destruction of a group of cells, the greater will be the degree of paralysis in the muscle. The greater the degree of paralysis in any muscle, the greater will be the degree of atrophy in the fibres of that muscle; so that any degree from slight to complete atrophy is possible. Inasmuch as the motor nerve to the muscle is merely a part of the motor cell in the cord it will be destroyed with its cell; hence an atrophy of the motor fibres in their passage through the antero-lateral column of the cord, and in the anterior root of the nerve, and in the nerve-trunk, to the muscle, is a part of the lesion of this disease.

Pathology.—The pathological change at the root of anterior poliomyelitis was first investigated by Cornil and Charcot. The specimens which they described were, however, obtained from chronic cases long after the onset of the disease; hence their description, while still valid for such older cases, does not represent the pathological process in its early stage. Within the past few years, however, autopsies have been obtained by various observers—among whom Drummond, Rissler, Goldschneider, Dauber, Redlich, Siemerling, and Trevelyan may be mentioned—upon cases which ended in death within a week or ten days, or a few months of the onset. The appearances as determined by these autopsies are as follows:—

The spinal meninges are congested, and here and there hæmorrhages of small extent are found in the dura and pia mater. The exterior appearance of the spinal cord is unchanged, but upon section the anterior portion of the gray matter, and frequently the central and posterior portions to a less extent, are found softened, so that they swell up above the cut surface, and are markedly red, with here and there small hæmorrhages and distended vessels. These changes are in some cases limited to one anterior horn; in some cases they extend through one entire enlargement of the cord; in others are found in both enlargements, or even throughout the cord. These changes are sometimes found more extensively disseminated than might be supposed from the clinical history of the case.

On *microscopical examination* there is found a marked hyperæmia of the tissue; all the blood-vessels are engorged and surrounded by exudation alike of serum, of leucocytes and of small cells. The serum fills the lymph spaces about the vessels and about the nerve-cells. The leucocytes infiltrate the tissues about the cells, and cluster around the cells. There is a great increase of small cells and nuclei in the neuroglia, which may be due to proliferation of the neuroglia cells or of the endothelial elements, or may be due to an emigration from the blood-vessels. This infiltration of the tissues with leucocytes and nuclei may be so intense as to obscure all other elements. Ruptured capillaries and small hæmorrhages are seen here and there. It is thus evident that the supporting substance (neuroglia) of the gray matter and the blood-vessels are involved in the inflammatory process.

The changes in the ganglion cells (motor neurons) of the cord are equally characteristic. These cells show great varieties of degenerative changes depending partly upon the severity of the case and partly upon the length of time the process has been going on in any one cell. The earliest change in the cell is the cloudy appearance of its protoplasm—an increased granular appearance obscuring the nucleus and leading to its deeper staining by the reagents. In the next stage of degeneration the protoplasm absorbs stains no longer; the nucleus is faint and the cell has lost its sharp outline, and some of its prolongations. Later still the cell appears changed into a swollen, shapeless, or spherical ball of matter, and its protoplasm is altered into a homogeneous unstained mass with vacuoles, or has become distinctly granular, in which case it stains deeply. At this stage the prolongations are entirely absent. The last state is one of shrinkage, the cell body being changed into a small deeply-stained mass hardly larger than its original nucleus. During the later stages leucocytes penetrate into the pericellular space and encroach upon the cell body. In the nerve-fibres, and in both the protoplasmic prolongations and neuraxon of the cells, similar processes of degeneration are found until a complete destruction of all the processes of the cell is brought about. During the first two stages of this process of inflammation in the cell an arrest may occur and repair be established; but the cells which have lost their processes are permanently damaged, and fall into atrophy.

The change in the interstitial tissue and in the ganglion cells is not in all cases parallel in degree. Some cases have been observed in which the cellular degeneration was attended by few or no changes in the vessels and interstitial tissue. If the case be examined some weeks or months after the onset the vascular changes are no longer evident; the serous exudation has been absorbed; there are no longer leucocytes and cells within the interstitial tissues, wherein only an atrophied rarefied neuroglia is left, containing a few normal cells and many nuclei, the relics of degenerated cells. In some parts the anterior horn may be changed even into true sclerotic tissue; and here and there a small cavity may be found within this sclerotic mass. In some cases the apparent change in the basis substance and neuroglia is very slight, and the only change is an atrophy of the ganglion cells such as Charcot described. The degree in which various groups of cells are affected varies greatly at different levels or even at the same level; so that some groups appear to be affected while others escape.

The result of this atrophy of the cells and surrounding tissue is a gradual shrinking in the entire size of the anterior horn; and as the nerve-fibres arising from these cells pass into the anterior and antero-lateral columns, and into the anterior spinal nerves, there is an accompanying atrophy in these columns and in these nerves. Many of the cells in the anterior horn are association cells with neuraxons which pass only to other levels of the cord through the antero-lateral columns; and these also suffer in the lesion. Hence in cords examined late in the disease there is a manifest deformity of the entire enlargement affected; and on section there is a decided shrinkage and apparent sclerosis of the columns around the anterior horn. This condition is shown in Fig. 19.

Considerable discussion has arisen on the exact origin of this inflammatory process, and two opposing hypotheses are current regarding it. The majority of recent observers believe that in anterior poliomyelitis we have an acute inflammatory process, similar to that in general myelitis but limited to the domain of the anterior spinal arteries; and that the atrophy of the spinal cells is the terminal stage of this process of general inflammation. A few observers (von Kahlden, for instance) still believe that there are cases in which the disease is a degeneration limited exclusively to the anterior cells, and not accompanied by any general inflammatory process; and these observers still maintain that the original description of Charcot is correct.

A hæmorrhage into the anterior horn, or a thrombosis of one of the branches of the anterior spinal artery, has been supposed to be the lesion in the cases which begin suddenly without fever; but although this hypothesis is likely enough, no actual observations can be found to support it. It has been shown by Allbutt and others that slight hæmorrhages occur in the spinal cord in children, either spontaneously or after slight injuries. The exact pathology of these cases is still unknown.

As a consequence of the spinal lesion there is a degeneration and atrophy of the anterior nerve-roots and of the motor fibres in the nerves of the body proceeding from the segment affected by the lesion. There is also a granular degeneration of the muscle fibres in the early stage; and finally the muscles are changed into fibrous bands, infiltrated in some cases with fat. Mention must also be made of the smaller size of the bones in the affected limb. This is rather an arrest of growth than a pathological state. Occasionally a true multiple neuritis is associated with a poliomyelitis (11), both due to the same cause.

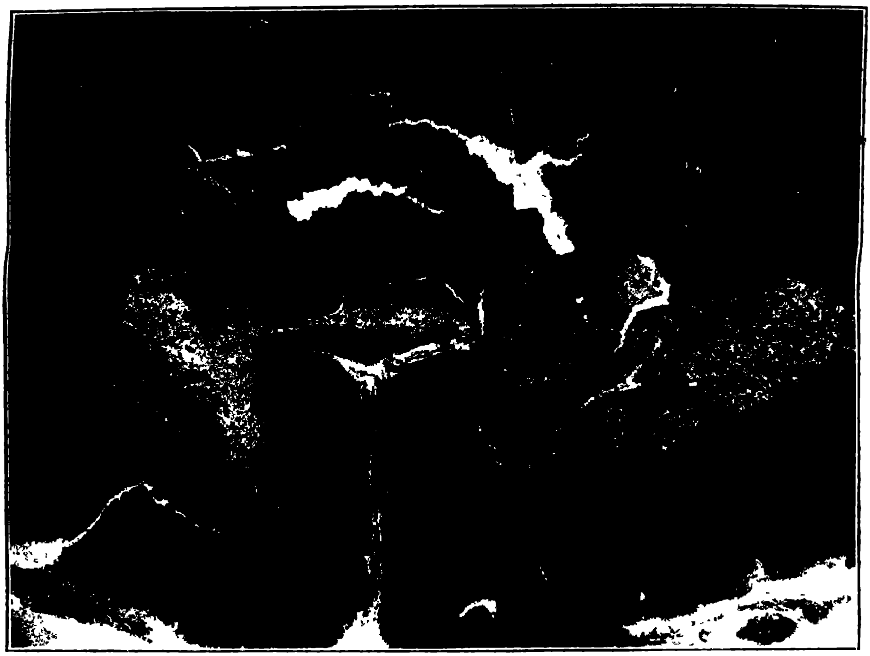

FIG. 19.—Section through the seventh cervical segment of the cord from a case of anterior poliomyelitis affecting the right side. The shrinkage of the entire right horn and the mass of sclerotic scar tissue occupying its centre are evident. All groups of cells excepting the lateral group have been destroyed.

Etiology.—Anterior poliomyelitis occurs with about equal frequency in the two sexes. Hereditary influences play no part in its causation. It is essentially a disease of infancy, although it occurs at all ages of life; cases have been noted in adolescence, in adult life, and even in old age. The following table (Table III.) demonstrates that the

TABLE III.—Age of Onset in Anterior Poliomyelitis.

	Years.									
	1	2	3	4	5	6	7	8	9	10
Seeligmüller	20	25	18	1	1	2	...	...	...	...
Galbraith	17	38	15	4	1	...	...	...	...	...
Sinkler	44	92	55	29	9	2	3	6	0	3
Gowers	21	21	25	9	17	4	2	6	4	...
Starr	16	38	27	9	10	4	2	2	4	3
Total	118	214	140	52	38	12	7	14	8	6

majority of cases occur during the second and third years of life, and that the disease is rather rare after the age of six. It has been supposed by some authors that cases of congenital club-foot are due to the occurrence of anterior poliomyelitis in utero; and it is certain that such cases are usually associated with a congenital lesion of the sacral region of the cord. This lesion, however, is usually due to a defect of development rather than to a vascular disease; hence the assertion that anterior poliomyelitis may occur in utero is open to doubt. That the disease may occur very early in life is confirmed by the case recorded by Duchenne, in which the child was but twelve days old; and Sinkler has recorded a case in which the child was six weeks old. The youngest patient in my own records was of the age of five months.

The season of the year appears to have a direct relation to the occurrence of infantile paralysis. Dr. Barlow called attention to this fact in his monograph in 1878, and it has been noticed by Sinkler in his numerous papers. Sir W. Gowers also confirms the statement. The preceding table shows the month of onset in the cases recorded by these authors, and in my own cases. It will be noticed that (Table IV.) the vast majority

TABLE IV.—Month of Onset in Anterior Poliomyelitis.

	Barlow.	Sinkler.	Gowers.	Starr.	Total.
January	1	4	1	2	8
February	0	3	1	1	5
March	4	9	1	6	20
April	2	4	1	2	9
May	4	10	1	3	18
June	5	27	11	6	49
July	16	52	13	16	97
August	11	65	13	27	116
September	4	29	15	17	65
October	3	25	6	8	42
November	1	4	2	4	11
December	2	3	5	2	12
	53	235	70	94	452

[illegible] cases occur in the months of June, July, August, and September; that is, during the months of greatest heat in England and in America.

The fact that this disease has occurred in epidemic form has thrown considerable light upon the etiology; and has made it extremely probable that it is to be regarded, at least in some cases, as an acute infectious disease. Colmer was the first to record the occurrence of the disease in epidemic form; for, in describing a case of this kind, he noted the fact that the parents of the child remarked that in the village in which they lived, eight or ten other children had been attacked with similar symptoms within a period of three months.

In a communication to the Society of Medical Science in Lyons, in 1887, Cordier announced that in the course of the months of June and July 1885 he had seen thirteen cases of infantile paralysis in the town of Sainte Foye l'Argentière, a town of a population of 1500 persons, where in other years the disease had been extremely rare. In 1890, Leegard reported that in the little village of Mundal in Norway eight cases of infantile paralysis had been seen between the last of July and the first of September; the disease never having been seen in the town before. Medin (12), of Stockholm, also describes an epidemic of the disease in Sweden. In 1888 he saw during the spring five cases; but during the summer the number increased rapidly, so that between the 1st of August and the 1st of November forty-four cases had been observed. Necropsies were made in some of these cases, which proved the disease to be a pure anterior poliomyelitis. Medin records the fact that in the town of Umea in 1881 Bergenholtz had observed a little epidemic of thirteen cases. The most extensive epidemic of the disease ever known was observed by Caverly in 1895 in Rutland, Vermont. Through his courtesy I examined a number of his cases during this epidemic. The epidemic occurred between the 20th of July and the 20th of September 1895, in a broad valley within a radius of twelve miles from the city of Rutland. Isolated cases within a radius of fifty miles were observed during that summer with unusual frequency. One hundred and forty cases of the disease occurred, of all grades of severity; and, though they chiefly appeared in infants and children, adults were not exempt. In many cases the atrophic paralysis was attended by pains; but in none were anæsthesia, bedsores, or permanent bladder and rectal disturbances produced. It was easily possible to exclude epidemic cerebro-spinal meningitis; and the resulting permanent palsies exactly resembled those left after atrophic paralysis. In the majority of cases the onset was acutely febrile, with marked constitutional disturbances. The epidemic ceased after the 20th of September. There has been no recurrence. Unfortunately no autopsies were obtained. Pieraccini observed seven cases occurring within a few weeks in July and August 1895 in a small village near Florence (Italy), where at that time an epidemic of whooping-cough was prevailing.

These facts prove beyond question that the disease may occur in the form of an epidemic.

Another indication of the infectious origin of infantile paralysis is its frequent occurrence in connection with other infectious diseases, or subsequent to them. Among my own cases, diphtheria, meningitis, measles, pneumonia, scarlet fever and acute malarial poisoning were noted as having occurred in a number of cases just prior to the onset of the disease. Gowers, it is true, questions the relationship between its occurrence and that of other acute febrile diseases, but the coincidence has been observed by too many different authors to be merely accidental. Exposure to cold or to sudden check of perspiration has been alleged as a proximate cause. I have seen several cases which have arisen in boys after swimming in very cold water for too long a time, and it is not unlikely that the frequency of its appearance in summer is due to occasional chill. Traumatism has also been regarded an immediate cause, and several cases are on record where children have been paralysed after a fall or a blow on the back (1). Inasmuch as we have already seen that vascular disturbances are at the basis of the affection in many cases, there is every reason to believe that traumatism may be a factor in the causation of the affection. Whether the functional hyperæmia consequent upon the increased activity in the lumbar region of the cord at a time of life when the child is learning to walk may account for the frequent occurrence of paralysis in the legs at that time, is an open question; but if hæmorrhage or thrombosis of the spinal vessels be accepted as the fundamental pathological condition in the non-febrile cases this factor must not be disregarded.

Diagnosis.—There is no difficulty in recognising the disease; indeed, it is hardly likely to be mistaken for anything else. Occasionally a child attacked with acute articular rheumatism, on account of the pain in the joints, is unwilling to move the limbs, and thus may be thought to be paralysed. A careful examination will soon demonstrate the true condition; for acute rheumatism never causes any atrophy or paralysis, and the local tenderness in the joints, the sweating, and the lack of coldness of the limbs may also aid in the diagnosis. Rachitis, sometimes caused among children living in healthy and comfortable surroundings by the use of artificial patent foods containing an excess of sugar, may appear with a sudden febrile onset, and much pain and tenderness in the limbs and unwillingness to move. But the child is not really paralysed; and the state of its bones, the general condition and the sweating, as well as the lack of limitation of the pain and immobility to one or two limbs will prevent this disease from being mistaken for infantile paralysis. In some cases of anterior poliomyelitis considerable pain is felt in the limbs, and some tenderness of the surface and of the muscles. The existence of pain during the first two days of the disease occasionally leads to mistakes in diagnosis. Thus Marsh records the case of a child, five years of age, who was suddenly attacked with pain in the left leg extending down the thigh to the knee. The limb was flexed, abducted and rotated outward, and any movement was painful; hence the case was recorded as acute hip disease, but a closer examination showed the hip-joint to be quite freely movable; and after two days, when the pain had passed away, the case was found to be one of infantile paralysis. The fever and general constitutional disturbances present at the onset had obscured the diagnosis. It has been suggested that when pain is severe a neuritis accompanies the *poliomyelitis. The existence of pain alone is not a* sufficient reason to warrant this diagnosis, inasmuch as the more recent pathology indicates that in the early stages there is a congestion of the gray matter of the cord which might be sufficient to explain the pain. If, however, the pain continue, and tenderness comes on in the muscles and nerves, it is probable that a neuritis due to the same infectious agent has set in. It is to be remembered that polyneuritis is usually a disease affecting the extremities symmetrically, and causing drop-wrist and drop-foot; that the distal parts are more severely paralysed than the proximal parts of the extremities; that there is no such selection of muscles paralysed as in poliomyelitis; and that there are usually sensory disturbances of a permanent nature—anæsthesia and analgesia or ataxia—in addition to the pain and tenderness along the nerves: hence in the stage of acute onset a polyneuritis should not be confounded with a poliomyelitis. When polyneuritis accompanies poliomyelitis, the clinical picture will be made up of a combination of these symptoms.

A localised neuritis of the brachial plexus (Erb's paralysis) causing paralysis of the deltoid, biceps, coraco-brachialis and supinator longus is not uncommon in infants, and might be mistaken for infantile palsy. The history of injury during delivery and the local anæsthesia in the distribution of the circumflex nerve will, however, correct the mistake. Such cases usually end in recovery.

Prognosis.—The prognosis in anterior poliomyelitis is always grave. Patients do not often die of the affection, but they rarely escape a permanent paralysis in some part of the body. It is true that in the majority of cases the original paralysis subsides, so that there is apparent improvement to a considerable degree. Thus a patient who has originally been paralysed in both legs may entirely recover the power in one leg, and may be left with a condition of paralysis in the peronei or in the anterior tibial group of the other leg; so that the final condition is very much less severe than that of the onset. As a rule the limb that is affected never entirely regains its power, and usually shows some atrophy and shortening; the growth of a limb is hampered by the existence of the disease, and hence in a growing child the unaffected limb outgrows the other. It is thought that an electrical examination two or three weeks after the onset of the disease will afford some ground for a prognosis; that the muscles which respond to the faradic current three weeks after the onset of the disease will eventually recover, while those that fail to respond to this current at that time will always be somewhat impaired. The loss of faradic reaction, however, is not an indication that these muscles will be totally paralysed, since the faradic reaction has been known to return in a muscle a year after it has been lost; yet such a muscle never completely recovers its size or power. The prognosis is much better in the cases which begin with fever than in those which do not.

Treatment.—The treatment of infantile spinal paralysis in the acute stage consists in keeping the child quiet in bed, and applying a mild form of counter-irritation along the spine; this is best done by a paste consisting of mustard one part and flour three parts, applied in a poultice along the back and removed as soon as the skin is reddened and then again renewed; so that for at least a week there shall be continual counter-irritation without the discomfort of a blister; or dry cupping along the spine may be applied frequently. Frequent sponging with alcohol and cool water is indicated in the cases in which the temperature is above 101°, but phenacetin or antipyrin are not to be used unless the temperature reach 103° F. There is some advantage to be gained from the internal use of ergot or of iodide of potash in the early stage, and moderate doses of salicylate of soda or of quinine may be used. If the child is in much pain or has convulsions, moderate doses of bromide, with or without codeia, may be employed as a symptomatic remedy. The general treatment of febrile conditions, a light diet, and laxatives are not to be neglected. Rest in a prone position in bed is better than constant lying upon the back.

When the acute stage has passed over, there is little to be done during the first week excepting to nourish the child well and keep the paralysed limb warm. Iodide of potash in three to five grain doses may be administered three times a day.

When the paralysis begins to subside spontaneously it is well to administer strychnine in full dose—$\frac{1}{30}$ of a grain three times a day for a child of three years of age. This remedy is best given at intervals, not continuously; it is my rule to use it for a week and then intermit it for three days. The condition of mechanical irritability in unparalysed muscles, as determined by percussion with a hammer, is a good indication of the degree of the effect produced by the strychnine, and the strychnine may be increased up to a definite point of increase in this irritation. It is to be remembered, however, that twitching of the limbs and stiffness of the back, usually indicative of strychnine effects, are not to be expected in infantile paralysis where the muscles are paralysed. Whether general tonics, such as cod-liver oil, hypophosphites or arsenic, have any effect of a favourable kind may be left to the judgment of the physician in each individual case. The most important indication during the stage of regression is to preserve the nutrition and function of the paralysed muscles; and this is to be attained by skilful massage, by hydrotherapy, or by the use of electricity.

Massage is of the utmost importance in these cases and should be given once or twice a day with care, combined with such attempts at active movement as the child is able to make. Among the poorer classes it is well to instruct the mother how to do this, so that it should be given with persistence. The massage should not be of the hardest kind, and yet should be sufficient to stimulate the circulation in the limbs, and to promote the lymphatic and venous flow.

Next to massage *mechanical devices* which shall induce the child to make use of the weakened limb are to be employed. A household gymnasium, adapted to the individual case, can easily be devised by the physician; and, if such exercises are made a kind of play for the child, much good will be derived from his own efforts.

Hydrotherapy has also an important use. The general circulation in the cold and flabby limb is aided by warm baths, and it is my habit to order these children to play in warm water (temperature 98° F.) for half an hour twice daily. This warm bath may be followed by a cooler sponging and brisk rubbing, but cold water should not be employed for these children as the temperature of the paralysed limb is always below that of health. Proper protection of palsied limbs by extra flannel clothing is always advisable.

Electricity is a valuable agent in the treatment of infantile paralysis, but a clear statement of its use should be made by the physician to the family. Electricity has no influence whatever upon the course of the disease. It does not affect the lesion in the spinal cord—either to decrease the hyperæmia, or to increase the nutrition of the nerve-centres. Applications of galvanism to the spine are therefore absolutely useless; but applications to the muscles may be of distinct service in two different ways:—first, by causing their contraction, and thus exercising them when voluntary exercise is impossible; and, secondly, by promoting those chemical changes in the muscle which are essential to growth and nutrition.

Examination of any case will reveal a certain number of muscles in the paralysed limb which respond to faradism. These muscles will eventually recover entirely, yet the tone and strength of the muscle should be kept up during the period of improvement by means of exercise with either the faradic or galvanic current. It is well proven that, as exercise of a healthy arm will markedly increase the size of the biceps muscle, so the application of faradism regularly to a susceptible muscle will increase the size of this muscle; hence to the weakened muscles which still respond to faradism an application of the faradic current for about ten minutes once or twice a day will be of service. The majority of the paralysed muscles do not, however, respond to faradism, and it is time wasted to apply the faradic current to these muscles, for they do respond, as a rule, to interrupted currents, the positive pole being placed over the muscle and the negative upon the limb at a short distance above. The interruptions should be made by an electrode held in the hand, and provided with a finger-key; and each muscle should be treated for about three minutes daily—fifty to sixty interruptions a minute being made by the finger. The strength used should be the least which will secure contraction in the muscle. When interruptions of the current do not produce a prompt response, alternation of the current may be employed by reversing the current rapidity by means of the pole changer on the battery. It is to be remembered that in this disease the application of electricity is more painful than in health. It is also to be remembered, in applying electricity to children, that their confidence must be gained; if they are frightened at the first application subsequent treatment will be a continual struggle. It is my custom, therefore, to begin a course of electrical treatment to a child by several applications of the sponges and electrodes while no current is passing, thus accustoming the child to the apparatus and gaining its confidence. After two or three such applications a weak current may be used, and then day by day its strength may be increased, until by the end of ten days the necessary strength is attained. In this way a daily struggle, with unsatisfactory and probably useless applications, can be avoided, and the parent's consent obtained to a course of treatment to which they would eventually object if every application meant a struggle. Any intelligent mother or nurse can be taught to give the galvanism or faradism to a child in this manner, and it is best to interest the attendant in the treatment from the beginning, and to instruct her carefully, so that within a week the treatment can be left entirely in her hands. Such an application of electricity is to be made daily, or twice a day for two or three years. Spontaneous recovery will have reached its best at the end of the first year, but even after this time these muscles may be brought into a condition of hypertrophy by means of continued exercise. When, however, a child is quite able with some force to move voluntarily any paralysed muscle, it is far better to rely upon voluntary exercises than upon electric applications. If at the end of a year no effect is obtained in a muscle from massage, bathing, and electricity, there is no use in continuing the treatment of that muscle, as it will never recover; its nerve-cells are entirely destroyed

The use of *mechanical apparatus* plays a great part in the treatment of infantile paralysis in the chronic stage. It is to be remembered that many weak muscles can do their work only when the limb is placed in an advantageous position, or when they are assisted in their action. Many of the muscles have, as part of their function, to keep the joints in place, and this part can be supplied by properly adjusted braces; hence an apparatus may enable the child to use a muscle or move a joint which it could not do if the joint were unsupported. Again the result of paralysis of one group of muscles is to allow the joint to be bent by its opponent, or to yield to the influence of gravitation; hence, if a brace be not applied early to correct this tendency, the paralysis is often followed by deformity. There is no disease in which orthopædic apparatus is of more service than in infantile paralysis, and it cannot be applied too early, as it may prevent the development of contractures and of deformities. There is no stage in which it is too late to fix a brace; for even when these deformities have occurred tenotomy may be employed to straighten and adjust a joint, and then the limb can be fixed by the brace in a proper position. But every case has to be treated skilfully in accordance with its own peculiarities, and the ready-made braces of the shops are often worse than none. A special apparatus for each case must be fitted under the direction of an orthopædic surgeon; and it is to be remembered that in a growing child such apparatus must be constantly readjusted, its length and size being changed from month to month in accordance with the development of the limb.

In many cases of deformity, where there is a strong contracture of a fairly healthy muscle overcoming the weak paralysed muscle, the question of *tenotomy* will arise. Tenotomy will of course result temporarily in a replacement of the deformed limb to its natural position; but, unless the joint can afterwards be held by a brace in a proper position, tenotomy alone will be of no permanent service. Hence, in some cases, tenotomy is only to be regarded as a preliminary to the proper application of apparatus. Apparatus has also been devised (especially in the treatment of infantile paralysis of the hands) by means of which weakened muscles may be reinforced by elastic bands so applied as to take the place of the paralysed muscle. Thus a drop-wrist or a paralysis of the extensors of one side of the wrist can be somewhat relieved by a series of elastic bands attached to the finger-tips, or to rings and to the elbow, and running through a bracelet at the wrist. Such devices, however, are usually discarded after a time, as they are more cumbersome than useful. Apparatus is especially applicable to spinal curvature of the paralytic kind; and in any case in which the body or back muscles are involved at the onset, it is well for the child to wear a brace in order to prevent the establishment of some form of curvature.

It has been proposed to attach a portion of the tendons of certain healthy muscles to the severed tendons of paralysed muscles about the ankle, knee, wrist, and elbow, in order that the healthy muscle may be made to do the work of the muscle which is paralysed, and a few successful attempts in this direction have been reported. I have seen one case in which the result was excellent, a portion of the tendon of the post-tibial muscle being attached to the tendon of the peroneus longus, whereby all weakness and deformity were corrected.

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POLIOMYELITIS ANTERIOR CHRONICA

UNDER this name will be included the chronic degenerative diseases which involve the cells of the anterior horns of the spinal cord, and in association with these, under Bulbar paralyses, will be described the chronic degenerative diseases of the representatives of these cells in the medulla oblongata, namely, the nuclei of the motor cranial nerves.

The name poliomyelitis anterior chronica expresses a chronic inflammation limited to the anterior part of the gray matter of the spinal cord; but the name does not really express the disease fully or correctly, for in the first place the disease is a chronic progressive degeneration, and, secondly, the morbid process may affect the lateral columns as well as the gray matter.

According to recent investigations the so-called motor system consists of two chief parts—(*a*) A pyramidal cell in the so-called motor area of the cerebral cortex with its axis-cylinder process, which is prolonged through the internal capsule, the pyramidal tract, the decussation of the pyramids, and the lateral column of the opposite half of the cord to the anterior horn, where it breaks up into ramifications in the neighbourhood of a cell in the anterior horn; (*b*) a large multipolar cell in the anterior horn which sends out its axis-cylinder process through the anterior root to a particular muscle fibre. We have, therefore, two classes of diseases corresponding to the affections of one or both of these two parts. When the anterior horn cell, with its prolongation to the muscle, is affected, we have the disease named Progressive spinal muscular atrophy; and when in addition the cortical cell and its prolongation are concerned, the disease receives the name of Amyotrophic lateral sclerosis (Charcot). The two diseases are described separately (see p. 176).

Although chronic bulbar palsies are manifestations in the pons and medulla of the same process which takes place in the spinal cord, they will be described more conveniently with bulbar paralyses.

PROGRESSIVE SPINAL MUSCULAR ATROPHY. — (Progressive myelopathy, wasting palsy).—This disease is characterised, clinically, by gradual wasting of the muscles of the limbs in definite groups, by fibrillar tremors, by the reaction of degeneration to electrical testing, and by the absence of any change in sensation; pathologically, by gradual degeneration of the ganglion cells of the anterior horns of the spinal cord.

History. — The first cases recorded were those described by Sir Charles Bell in 1836, who attributed the condition to a lesion of the nerves, and also by Darwall, who pointed out that the atrophy came on after carrying heavy weights. But the first definition of a distinct type of atrophy affecting particular groups of muscles was made by Aran in 1850, who described eleven cases, and noted that the small muscles of the hand were first attacked, and the flexors of the wrist and fingers before the extensors; also that the biceps and deltoids were wasted while the triceps was intact. Fibrillar contractions also were then observed, and Aran considered the disease to be in the muscles themselves, and not in the nerves. He gave the name of Atrophie musculaire progressive to the disease.

Duchenne, in 1853, published a memoir on these cases; and in the same year Cruveilhier described three cases with necropsies. In two he found nothing abnormal in the nervous system; but in the third atrophy of the anterior roots was found.

In 1867 Lockhart Clarke first found diminution in size and changes in the cells of the anterior horns, and that the whole of the gray substance was replaced by a "cylinder of morbid matter."

Charcot's writings have done much to connect the wasting of the muscles with the atrophy of the anterior horn cells. At first all muscular atrophies were thought to be due to lesions of nerves, then, until Clarke's discovery in the anterior horns, they were attributed to lesions of the muscles themselves. Of late years the muscular atrophies have been separated into those depending on a lesion of the muscles themselves (myopathies), and those depending on a lesion of the spinal cord (myelopathies), the latter of which we are now considering.

Causes.—*Age.*—The disease is one of adult life: it rarely begins before the age of twenty-five, and it occurs up to fifty; but a case has been published by Thomson and Bruce in a child, aged 1½ years, presenting some of the symptoms of progressive muscular atrophy, in which atrophy of the cells of the anterior horns was found after death. As a rule, cases of muscular atrophy occurring in children are those of idiopathic muscular atrophy.

Sex.—Males are more affected than females.

Heredity.—In a certain number of cases—about half—there is a history of some diseases of the nervous system in other members of the family; but there is not, as a rule, that tendency of several members of one family to be attacked which we may see in the idiopathic form: exceptions to this rule are given on page 213.

Proximate causes.—Getting wet through and catching cold, and injury to the back are alleged as causes in some cases; and although it is often difficult to accept the former as a cause, injuries are more likely to have such effect, especially when, as in some cases, the atrophy begins in the injured limb; in other instances the injury has been a blow on the back or a general concussion of the whole nervous system.

Syphilis has been considered a cause, and in one case which I saw there was a distinct history of syphilis; but treatment directed against this disease did not improve the condition. If syphilis play any part, it is probably by altering the nutrition of the nervous system and disposing it to degeneration, as in locomotor ataxy.

Excessive worry and anxiety have been given as immediate causes, and sexual excesses also. In many cases no cause can be guessed at.

Other organs.—The other organs of the body present no evidence of disease associated with the condition of the nervous system; but with patches of sclerosis occurring in the medulla it is not surprising that palpitation and dyspnœa should be noticed. So too the urine sometimes contains sugar, which disorder has also been found associated with patches of sclerosis in the medulla.

Course and duration.—P. Marie has distinguished four modes of progression in this disease: 1. A chronic progressive course; 2. A chronic course with sudden intercurrent attacks of disturbance of vision, apoplectiform seizures, hemiplegia, and the like; 3. A chronic remitting course; 4. Increasing improvement or apparent cure.

From what has already been said in describing the manifestations of the disease, it will be obvious that the third of these modes is the most common; while, if we exclude the fourth group, which probably does not deserve consideration except as part of the third, the least common mode is that included under his second group.

We have seen how common it is to meet with periods of arrest, or even of improvement amounting in some cases perhaps to apparent cure. And we have also seen how such remissions are followed, after a variable time, by relapses; several such improvements and relapses possibly occurring in the course of the same case. That no hard and fast line can be drawn between such cases characteristic of Marie's third group and his fourth is obvious in that although he includes in the fourth group only the cases in which there is apparent cure, we have seen abundant evidence that, after long periods of arrest, exacerbations occur; and probably no case of true multiple sclerosis ever ends in recovery. The chronic progressive mode of Marie's first group is characteristic of the classical type of the disease, and is next in frequency of occurrence to the chronic remitting mode. Charcot has described three stages in the course of this chronic progressive mode: the first from the time of onset until the spastic paralysis and tremors are established; the second in which the patient is more or less confined to bed, the spastic paralysis has gone on to contracture, and all the symptoms of the first stage are further advanced;—this is a stage of the disease which may last for many years, during which the nervous symptoms do not progress materially, and the patient's nutrition is well maintained; the third stage is characterised by general nutritional disturbances, loss of appetite, emaciation, and tendency to decubitus; the psychical disturbances are far advanced, the speech defect is more marked, the sphincters are paralysed, cystitis sets in, bedsores form in neglected cases, and death results from pyæmia.

Whatever the mode of the disease, this final stage is common to most cases of disseminate sclerosis. Some intercurrent affection, however, such as pneumonia, pleurisy, or phthisis, may prove fatal; in some cases, again, death is caused by bulbar paralysis consequent on patches of sclerosis located in this part.

Nothing is more difficult than to fix the duration of the disease; the diversity is too great. All that can be said is that the duration is to be reckoned by years; and that, according to Charcot, whose statement has been confirmed by subsequent writers, patients in whom "spinal" symptoms alone are present may live for twenty years or more after the first manifestations of the disease.

Morbid anatomy.—The patches of sclerosis are irregularly distributed throughout the nervous system in the most random manner possible: there being no part which is not liable to be so affected, though some parts appear to be especially prone to invasion. That the disease is a general one, at any rate so far as the central nervous system is concerned, there can be no question; and, accordingly, the view formerly held that a cerebral as distinguished from the spinal form of the affection can be recognised pathologically as well as clinically is no longer tenable. Although the clinical manifestations of the disease may point to the brain or spinal cord being separately affected, it is highly improbable that a careful examination of the nervous system after death, macroscopically and microscopically, will fail to reveal unequivocal evidences of affection of all parts of the central nervous system at least; whether any concomitant affection of the peripheral nervous system be present or not. As we have already seen, of the apparently isolated affections of a particular region of the nervous system, the spinal form is the more frequent; it is easy to understand therefore how patches of sclerosis in the brain, too small to give rise to symptoms, may readily be overlooked even after death, unless great care be exercised in the examination of the organs. On the other hand, in rare instances where the symptoms point to affection of the brain alone, little difficulty can be experienced in detecting evidences of concomitant affection of the spinal cord; that is, if proper opportunity is found for the study of the morbid anatomy of the case.

Charcot, while recognising a pure spinal form of the affection, admitted that in the cerebral form the possibility of areas of sclerosis being found also in the spinal cord could not be excluded; and Erb believes that a purely cerebral or spinal form of the disease is very rare.

An exception to the rule appears to be forthcoming in a case of disseminate sclerosis, recently published by Probst, in which, with patches of sclerosis in the pons and medulla, there existed no such sclerotic areas in the spinal cord: there was, however, a systemic degeneration of the crossed and direct pyramidal tracts; the pyramidal fibres, from the proximal end of the pons to the lumbar region of the spinal cord, being degenerated. There was also a less pronounced degeneration of the postero-internal columns, extending from the upper part of the thoracic region of the cord to the termination of the tracts in the nucleus of the funiculus gracilis on each side. As I have said, although in so far as the nervous system is concerned the disease is a general one, it appears, nevertheless, to be more prone to invade certain regions than others. If, in the first place, it appears to affect the central nervous system, as opposed to the peripheral, more commonly and in greater degree, we must remember that even this statement, based, as it is, on past observations, is one which may have to be considerably modified in the light of future researches; for the peripheral nervous system in cases of disseminate sclerosis has not been examined with anything like the regularity and care that has been bestowed upon the central nervous system.

Most observers have recognised certain *seats of election* in the disease. According to Sir William Gowers, the centrum ovale of the cerebral hemispheres is a frequent seat, whereas few patches occur in the cerebellum. Erb finds the floor of the fourth ventricle, the pons, the walls of the lateral ventricles, the white substance of the cerebral hemispheres, and the white tracts of the spinal cord, to be the parts most commonly affected. Strümpell is of opinion that the patches of sclerosis occur specially in the white substance of the hemispheres, the walls of the lateral ventricles, and the corpus callosum; that they are somewhat plentiful in the pons; less so in the medulla; but very plentiful in the spinal cord, especially in its white substance. My own observations are in harmony with those of Strümpell, which, as may be seen by comparison, differ little from those of Erb. The former observer's statement, however, that the medulla is usually less affected than the pons and spinal cord, does not accord with what I have found in the cases that I have examined. I am in a position to corroborate the statements of those observers, including Sir William Gowers, who have found that, as a rule, few patches occur in the cerebellum; but my observations do not accord with those of Obersteiner, who finds that patches of sclerosis occur less commonly in the lumbar region of the spinal cord than at higher levels.

A further point of importance, with regard to the distribution of the sclerotic areas, is the degree in which the white matter is affected as compared with the gray. Since the time of Charcot, who was of opinion that the patches are rarely found in the cortex of the brain or cerebellum, most observers have expressed similar views—Sir William Gowers believes that the patches seldom invade the gray matter of the cortex, and that they never appear to begin in it. It is very striking, sometimes, to see how strictly limited the patches are to the white matter, at any rate as seen macroscopically, abutting on the surface gray matter both in the cerebrum and in the cerebellum, without actually invading it; but that in other instances the gray matter of the cortex is invaded there can be no question. Dr. E. W. Taylor finds no justification for the view that, in respect of the general distribution of the disease, there is any seat of election; he not only finds that the gray matter of the cortex of the cerebrum and cerebellum does not escape, but also that the patches may begin in the cortical gray matter, and be limited to it. This latter point I have not myself had an opportunity of observing; and, while fully agreeing with Taylor that the gray matter of the cerebrum and cerebellum does not escape, my observations certainly support the views of those who regard these parts as being less commonly affected than the white matter. A curious and interesting fact, one on which others have commented also, is that, in spite of this slighter tendency to affection of the surface gray matter of the cerebral hemispheres, the gray matter of the basal ganglia appears to be a favourite seat for the sclerotic process.

There seems to be no rule as regards the frequency of affection of the gray and white matter of the crus, pons, and medulla oblongata; and the view that the ventral aspect of the pons is more commonly affected than the dorsal is not supported by my observations. The cases I have examined show an irregular, random distribution in the crus, pons, and medulla, with no respect for gray matter as opposed to white; indeed, in so far as these observations go, the gray matter and mixed regions were possibly more affected than the purely white matter of the ventral aspect of these parts of the central nervous system. Buchwald and others thought that certain systems in the spinal cord are specially prone to participate; and Bourneville and Guérard noted a predominance of the patches in the symmetrical columns of the cord. However true these statements may be, the fact remains that the most varied pictures may be seen in the spinal cord as a result of the distribution of the sclerotic areas. I have seen a practically complete transverse lesion produced by a patch of sclerosis; an exceedingly small group of myelinated fibres alone remaining in one part of the periphery of the transverse section: in other instances an almost accurate hemisection had been similarly produced; in others but a quadrant of the sectional area had been involved, while in others again several patches were scattered indifferently through the transverse section. As far then as my observations go, they certainly accord with those of others who have found the patches of sclerosis most commonly affecting the white matter of the cord; but the gray matter by no means escapes, for patches of sclerosis may be found not only invading the gray matter, but, in certain regions, practically limited to it.

The degree to which *the peripheral nervous system* is affected in disseminate sclerosis is a question that has received comparatively scant attention; so that, beyond certain facts in connection with the cranial nerves and the spinal nerve-roots, we have very little definite information to guide us. Nearly all authors have noted affection of some of the cranial nerves. Bourneville and Guérard, while citing the observations of Cruveilhier, Skoda, Vulpian, Lionville, and Ordenstein—who found patches of sclerosis in the hypoglossal, vagus, glosso-pharyngeal, optic, olfactory, and oculo-motor nerves—believed that the cranial nerves generally escape. Jolly also found the cranial nerves unaffected; while Charcot was of opinion that the optic, olfactory, and fifth cranial nerves alone are affected. E. W. Taylor found participation of all the cranial nerves (with the exception of the olfactory, which was not examined) in two of his cases; either the nuclei, the central portion of the nerve from the nucleus to the surface of the pons or medulla, or some part of its peripheral course being diseased. In a third case he found the degeneration not so pronounced; but the roots or nuclei of most of the nerves were affected. Claus found the third, fourth, fifth, and twelfth cranial nerves most affected; the seventh and eighth but slightly so, while the olfactory and optic nerves were free from change. Similar escape of the olfactory and optic nerves, which are usually regarded as favourite seats of the morbid process, has been noted by Probst, who found the facial, vagus, hypoglossal, and motor part of the fifth cranial nerves affected.

Uhthoff, who has made a careful study of the optic nerves in disseminate sclerosis, finds that some of the patches met with in them are indistinguishable from those of a tabetic atrophy; otherwise the process is of an interstitial neuritic character. In all the cases examined by E. W. Taylor the optic chiasma was the seat of patches of sclerosis; and in two of them so pronounced was the change that scarcely any myelinated fibres were to be seen. Of two well-marked cases of disseminate sclerosis, which I have examined comparatively recently, the optic chiasma of one, while presenting patches of sclerosis, had much of its structure preserved; yet the optic tracts behind and the nerves in front were much more extensively destroyed: in the second case the chiasma and tracts were intact, and the peripheral portion of each nerve for a short distance behind the globe was affected; although a portion of the nerve next to the normal part contained no myelinated fibres, a few such fibres were to be seen in the portion of the nerve immediately behind the globe.

Charcot, Erb, E. W. Taylor, Probst, and others have noticed that *the spinal nerve-roots*, both motor and sensory, may be attacked; and Taylor in his cases found also an extensive degeneration of the nerves of the cauda equina; but, so far as I have been able to discover, neither this observer nor any other has made any systematic examination of the peripheral system of spinal nerves. My own observations in this direction have been too few and too fragmentary to be of sufficient value for publication at present.

The appearance of the islets of sclerosis is very characteristic, and, it appears to me, quite unlike that of any other morbid process met with in the nervous system. The patches are of a warm gray colour, or of a brownish or reddish gray; they are both darker and more translucent than the gray matter of the cerebral cortex. Sometimes they have quite a gelatinous appearance, and are more or less of this consistence on palpation, being distinctly soft; in other instances the patches, though somewhat glistening, are firm in consistence. The sclerotic areas are irregular in outline, and to the naked eye appear sharply delimited from the adjacent healthy nervous tissue; they vary in size from areas a few millimetres in diameter to others whose diameter may be measured in centimetres rather than millimetres. When seen on the external surface of some part of the central nervous system, such as the pons, they may be on a level with the surrounding tissues, or may be slightly more prominent; while as seen on cut section of some part they are usually on the same level as the immediate surrounding tissues, or are, it may be, slightly depressed below that level. They are to be seen both on the external surface of the spinal cord and on section, and are usually greater in vertical than in transverse extent.

The microscopical examination of affected parts of the central nervous system may quite corroborate the impression gained on macroscopic examination in regard to the definite limitation of the affected areas from the surrounding healthy tissues. Charcot was of opinion that an intermediate zone of slighter affection separates the healthy from the diseased parts; and, although this is true in the case of some of the patches, my observations quite accord with those of E. W. Taylor, who found, in the case of other patches, an abrupt transition from diseased to healthy tissue, without interposition of any zones of intermediate degrees. I have seen an area in which all the fibres were deprived of their myelin sheaths lying next to an area whose fibres had all their myelin sheaths preserved; the change from healthy to diseased tissue being quite abrupt. In my experience, even in the majority of instances where an intermediate zone exists, the transition from normal to abnormal, in the sense of complete absence of myelinated fibres, is very rapid. There is, however, this difference to be noted, that it is much more common to meet with abrupt delimitation in regard to the lateral limits of the patches when they affect long tracts of parallel fibres; whereas there is, as a rule, a much more gradual transition from the normal to profound alteration in so far as the ends of such areas of sclerosis are concerned. Thus in the spinal cord if a longitudinal section be made, and an area of sclerosis found situated in a tract of parallel fibres, the non-myelinated fibres at the periphery of such an area may be seen lying side by side with normally myelinated fibres; whereas the upper and lower limits of the sclerosed area may show no such abrupt limitation, the fibres showing various intermediate stages of degeneration between the point where they are devoid of their myelin sheaths and that where their myelin sheaths are perfectly intact.

There is one point at least with regard to the histological changes met with in disseminate sclerosis on which there is unanimity of opinion among observers: all are agreed that the nerve-fibres in the affected areas are deprived of their *myelin sheaths*. It is a striking fact that while sections prepared by the Weigert-Pal method show this complete absence of myelin sheath which, normally, takes on the hæmatoxylin stain, sections prepared by the Marchi method show but little evidence at the margins of the affected areas of the process of destruction going on in the myelin sheaths, such as might reasonably be anticipated if the process be progressive, even though chronic. So chronic and slow in progress is the disease in some instances that the absence of such evidence is not surprising; but I have met with one instance at least in which the clinical manifestations of the disease had been in existence for a period sufficiently short to warrant the expectation that this evidence of destruction of myelin would be found, yet there was singularly little confirmation of the conjecture.

Where transition stages of the destructive process are met with, the nerve-fibres, instead of being completely denuded of their myelin sheaths, present various degrees of alteration of the same; the sheath may be swollen more or less uniformly, or in an irregular manner; knobby swellings of it may alone remain attached to the axis-cylinders in parts; or there may be no evidences of acute destruction of the myelin sheath, but rather an atrophy of it, a narrow ring only of myelin remaining around the axis-cylinder. Here and there granular cells and corpora amylacea may sometimes be seen.

The degree of resistance which the *axis-cylinders* offer to the destructive process is a curious feature of the disease. This has been insisted on by the different writers who have dealt with the subject from the time of Charcot to the present day. Areas in which there is a complete absence of myelinated fibres may, nevertheless, be occupied by naked axis-cylinders, apparently unaltered in character. This does not always obtain, however; for the axis-cylinders, though capable of resisting the destructive process, may in the end succumb like the other parts of the nerve elements. Popoff has recently expressed the view, and brought forward evidence to support his contention, that there is not in reality this supposed escape of the axis-cylinders in the destructive process of the nerve elements; but that, having been destroyed, they are capable of regeneration, and that it is to this capacity that the naked axis-cylinders owe their presence. One of the facts on which Popoff places most reliance is that, according to him, the ends of many of the axis-cylinders may be seen to be composed of a bunch of fibrils, just as is seen in the process of regeneration of peripheral nerves; but as yet this description stands unconfirmed by other observers; there has been nothing in the appearances in the cases I have examined to lead me to any such conclusion. Weigert, whose observations are based on his selective neuroglia stain, concludes that Popoff's supposed regenerated axis-cylinders are neuroglia fibrils.

The preservation of the axis-cylinders, in great degree at any rate, in disseminate sclerosis leads us next to consider what is perhaps the most extraordinary feature of the disease, certainly the most difficult of explanation; namely, that it is exceedingly rare to meet with any evidences of secondary degeneration of the nerve elements; and that when any such evidences are found, they are limited, as a rule, to a very small extent in the immediate neighbourhood of a sclerotic area. On examining, for instance, some of the affected parts of the spinal cord in which the morbid process has invaded one or other of the long systemic tracts, it seems incredible that no secondary degeneration of the fibres of the tract is to be found in regions remote from the sclerosed area; nevertheless, such is the case. The most reasonable explanation that has been offered for this absence of secondary degeneration of the nerve elements in disseminate sclerosis is that first suggested by Schultze, who attributed it to the preservation of the axis-cylinders, supposing that secondary degeneration only results when these are destroyed. Reasonable as this explanation is, it is not altogether satisfactory; for most observers are agreed that in the later stages of the morbid process destruction of the axis-cylinders does result; and this, it may be, in no mean proportion: yet even in such cases no secondary degeneration may be forthcoming. To explain this discrepancy, it has been urged that the destruction of the axis-cylinders does not take place in sufficient number to allow of the secondary degeneration being recognised—an argument which, while possibly justified where observations are based on the Weigert-Pal method of staining, cannot be regarded as satisfactory in so far as the Marchi method is concerned; for by the latter method isolated degenerated fibres can be picked out, and the presence of a collection of them is not required for the detection of the degeneration. Popoff's hypothesis with regard to regeneration of the axis-cylinders does not help us; as before regenerating they must be destroyed, and with their destruction we ought to find evidence of secondary degeneration, whether they are subsequently renewed or not. Yet there are very few recorded cases in which secondary degeneration has been established with certainty. Among those who have observed such degeneration are Jolly, Werdning, Babinsky, Redlich, and Rossolimo; but in cases of the kind the secondary degeneration has usually been traced but a very short distance from the sclerosed area. Schultze has recorded an instance in which a tract degeneration was established with probability in a case complicated by paralytic dementia. So Buss, too, found descending degeneration through the whole lumbar cord, and an ascending degeneration of Goll's column and the direct cerebellar tract from the level of the eighth cervical nerve-roots to the medulla oblongata; but such a state of things must be exceptionally rare. The case recorded by Probst, and already referred to, is remarkable in that, with areas of sclerosis in the pons and medulla and none in the spinal cord, there was not only degeneration of the pyramidal tracts, but also of the postero-internal columns in the upper part of the cord. Another notable feature in the case is that, in spite of other tracts in the pons and medulla being as much or even more affected than the pyramids, these fibres alone showed signs of secondary degeneration. This case, however, seems open to the interpretation that two independent diseases existed in the same patient; though Probst considers this not to be the case, and has adduced what he considers adequate proof in support of his contention. Most observers have found either no secondary degeneration at all, or degeneration limited to a very short distance from the sclerotic areas. I have seen sclerosis so situated in system tracts as to be mistaken for a tract degeneration by those not sufficiently familiar with such appearances to be able to judge of their real significance; so that only the results of experienced observers can be relied on with any degree of confidence in this matter.

Like the axis-cylinder, that portion of the neuron which comprises *the cell* is also unaffected till late in the course of the disease. Most authors state that such late affection of the ganglion cells does take place; but Koppen, Rossolimo, and others have found these cells normal. It is surprising how much sclerosis may be found in the immediate neighbourhood of such cells without the cell itself showing changes; even when examined by more recent and delicate methods, such as that introduced by Nissl, and variously modified since. On the other hand, that the cells are destroyed when the sclerosis is most intense in the gray matter appears to me beyond question. Charcot and Bourneville, and Guérard, all described a yellow degeneration of the cells; Sir William Gowers speaks of them as undergoing atrophy in the neighbourhood of sclerotic tissue; and Obersteiner considers that all the changes met with in the cells in myelitis may be present in this condition. Formann, Schuster, and Bielschowsky have also noted cell changes.

E. W. Taylor, while fully recognising that pigmentation of cells may have no pathological significance, more especially in old persons, is of opinion that, taking into consideration the age of his patients and the degree of pigmentation met with in two of the most advanced cases, the condition must in them be regarded as pathological. But for the preface to Taylor's observations, one would take any such results with considerable scepticism; as pronounced pigmentation of ganglion cells may be met with where they clearly have no pathological significance. The necessity for caution is further suggested by the fact that neither the axons nor dendrons of such cells showed any evidences of degenerative changes in his cases. Apart from this widespread change in the cells, however, Taylor states that he met with cells without nuclei in various stages of degeneration; and that, while the nerve-cells of the nuclei of the cranial nerves were mostly fully preserved, he established with certainty a diminution in the number of cells in the oculo-motor nucleus, especially on one side. In one case he found that the cells were preserved in areas of the cerebral cortex where sclerosis was present, but that where the sclerosis was very advanced distinct atrophy with diminution in the number of cells appeared. In his third case, in which the sclerotic process was not so advanced, no distinct cell changes were found, with the exception of a group of ventral horn cells in the cervical region of the spinal cord. Taylor, therefore, concludes that degeneration of the ganglion cells occurs, but only in an advanced stage of the process; for a long time they remain intact, and for the most part functioning, a fact that explains well why in disseminate sclerosis muscular atrophy and the reaction of degeneration are rare phenomena. In Probst's case the cells of the hypoglossal, vagus, facial, and trigeminal nuclei were affected by sclerotic patches, which compressed their destruction; while, in the absence of any sclerotic areas in the spinal cord, such changes as were seen in the ventral horn cells were regarded as comparable with the changes which are met with in these cells secondarily to cerebral lesions.

Marked increase of *the interstitial tissue* has been described by nearly all observers as forming part of the anatomical picture. A case recorded by Reinhold was quite exceptional in this respect, in that there was extensive disappearance of the nerve-sheaths and axis-cylinders without increase of the glia tissue, except in the centre of the patches where there was slight hyperplasia. As a rule there is marked proliferation of the interstitial tissue, the dense mass of fibrils being arranged longitudinally where the patches occur in association with any of the long tracts of the central nervous system; many small glia cells are also seen, forming a feltwork of interlacing fibrils when some part of the gray matter is affected. In the latter case, instead of the fibrils being arranged parallel to the nerve-fibres, they spread out in all directions from a centre where the tissue is most dense; and they may be seen insinuating themselves between the nerve elements at the periphery of the patches where the fibrillary meshwork is less dense; this change being accompanied by the presence of innumerable glia cells. Sometimes patches of increase of interstitial tissue are to be seen on the surface, and the pia may be adherent in such places; but there is nothing in such findings to support a view, formerly held, that the whole morbid process in this disease originates in the pia. The majority of the patches of sclerosis have no connection whatever with the pia, and can in no sense be regarded as depending on any morbid process engendered in this membrane. Moreover, the slight changes which may sometimes be seen in the pia connected with a patch of surface sclerosis are clearly secondary to the change in the cord, or other part of the nervous system concerned. Apart from such areas, the pia shows no evidence of any alteration in structure. Some observers have described a round cell proliferation as the earliest stage of the interstitial hyperplasia, such cells being described as especially plentiful in the neighbourhood of blood-vessels. Rindfleisch, Leubuscher, Leyden, and Chvostek noted proliferation of the cells in the neuroglia. Chvostek and Ribbert held that the leucocytes wander out of the vessels and then become changed into fixed cells. Förster also held that wandering leucocytes might become transformed into glia cells; so that originally wandering leucocytes were supposed to become fixed cells of connective tissue type, which subsequently develop into fibrils. In the morbid patches it is difficult to be certain how far glia fibres and glia cells are related to each other; in all probability, however, glia fibres spring from the cells. The precise appearances met with depend on the ages of the patches: thus in early patches little fibrillary structure is to be made out, the very fine fibre network being scarcely visible, and the chief change being a cell increase; in older patches the glia cells are more separated from each other, and the network of fibres is better seen. The oldest patches may have quite a homogeneous appearance; and in such patches Probst found, in addition to the small glia cells, larger spider cells (14 to 20 μ) with large nuclei; their processes forming a beautiful meshwork, the interspaces of which were homogeneous, with no ground substance to be seen; this description has been corroborated by Frommann, Hüber, and Redlich.

While changes occur in connection with *the blood-vessels* in a certain proportion of cases of disseminate sclerosis, such changes are not met with invariably; some cases show no departure from the normal in this respect. The vessel walls may be thickened to a variable degree; in some cases the increase in thickness is slight, while in others it is pronounced; but nuclear proliferation is usually slight. Moreover, the vessels are sometimes engorged, and a marked increase of the number of the smaller vessels may be seen, especially of the capillaries. In such cases white blood cells may escape from the vessels into the surrounding tissues, or small extravasations of blood are visible. The perivascular spaces are widened in many cases in which vascular changes are met with.

Rindfleisch first described affection of the vessels in disseminate sclerosis, and a great many observers have since met with similar changes; but others, among whom may be mentioned Leyden, Jolly, Buchwald, Hüber, and E. W. Taylor, have been unable to find any alteration in the vessels. In a case recorded by Probst, though there were no changes in the vessel walls, there was engorgement of the capillaries and increase of them in places, with widening of the perivascular space—conditions which have also been described by Leo, Kelp, Buchwald, Putzar, and others. Charcot, Guérard, and Frommann are among those who have found vascular changes in disseminate sclerosis; and, in addition to the more commonly described changes, they met with narrowing of the vessels. Ribbert, another author who has described changes in the vessels, found thrombus formations. Most observers have described thickening of the vessel walls, widening of the perivascular lymph space with a large number of leucocytes, and increase of nuclei of the vessel walls.

Pathology.—Leaving out of consideration those points in the pathogeny of the disease already sufficiently dealt with in discussing its causes, we have still to consider the origin of the morbid process. Various views have been advanced on this subject, and cogent arguments have been used in support of this or that hypothesis. The first questions are whether the morbid process is one which affects the nerve elements primarily, or whether the interstitial tissue suffers first? The explanation commonly accepted is that the process is primarily interstitial, consisting in a chronic inflammation; and that the myelin sheaths of the neurons suffer secondarily—a view that has been supported by Charcot, Erb, Gowers, Leyden, Wernicke, and others. Leyden has gone even farther in this direction, and has determined that a multiple sclerosis may be evolved from an acute myelitis; moreover, in their article in Nothnagel's *System of Medicine*, this observer and Goldscheider have treated disseminate sclerosis as a form of chronic myelitis. Bikeles also classes this disease as a myelitis.

The opposite view, which regards the disease as a primary affection of the myelin sheaths of the neurons with secondary hyperplasia of the interstitial tissues, owes its origin to Adamkiewicz, whose conclusions were based on the results of a safranin stain which is supposed to have a specific colour reaction for healthy and diseased myelin; but as the method is untrustworthy, the arguments based upon it are frail. A case, recorded by Reinhold, and already cited, may be interpreted on the lines of Adamkiewicz's view. The anatomical changes were quite unusual; as has already been said, in the presence of extensive disappearance of the myelin sheaths and axis-cylinders there was little or no increase of the glia tissue; and Reinhold regarded the case as an early one of an ischæmic or toxic degeneration, with much less of an inflammatory character. Hüber supports the view that the nerve elements suffer primarily, and that the sclerosis is secondary to the affection of the parenchyma. He considers that there is a simple degenerative destruction of the myelin sheath or of the whole nerve-fibre, and that the process is not inflammatory as we are accustomed to see inflammation in acute myelitis. Redlich holds similar views. Both of these observers could detect fresh patches where there was disappearance of the nerve-fibres in great masses without change anywhere in neighbouring interstitial tissue. Hüber's view has much to recommend it; moreover, a point which has oftentimes been suggested to me by a study of the morbid anatomy of this disease has not escaped his attention also; namely, that an interstitial proliferation, originated by a parenchymatous degeneration, may spread beyond the limits of the nerve elements primarily diseased, and may thus lead to secondary destruction of the nerve elements of contiguous parts. In this way the fact may possibly be explained that in the outlying districts of a patch of sclerosis the interstitial tissue change is often obviously in advance of the destructive process in the nerve elements. This may be seen more especially where the gray matter is invaded in any region; for under such circumstances, as has already been said, the interstitial tissue may be seen insinuating itself between nerve-cells wherein hitherto there had been no evidence of any deleterious effects on these parts of the neurons. The importance of giving due consideration to this possibility will be obvious when it is remembered that this very fact, that in the peripheral zones of sclerotic areas the interstitial process may oftentimes be seen much in advance of any destruction of the nerve elements, has been adduced as strong evidence that the whole morbid process is primarily interstitial; whereas, in reality, the process may be primarily parenchymatous, giving rise to proliferation of the interstitial tissue secondarily, while the hyperplasia thus started may in its turn lead to destruction of other nerve elements. According to this view, some of the nerve elements suffer primarily, while others suffer secondarily to an interstitial tissue proliferation originated by the primary degenerative changes in the one set of nerve elements.

The view that the morbid process is primarily parenchymatous accords well with the growing belief that the disease is toxic in origin; for the effects of toxins on the nervous system are as a rule first manifested on some part of the neuron itself, whose destruction leads to secondary proliferation changes in the interstitial tissues.

Another view held by some is that the process is primarily vascular, and that the vessel changes lead secondarily to the other morbid conditions. This interpretation of the morbid picture was that adopted by Rindfleisch as long ago as 1863; and it has since found favour with different observers, including Ribbert, Buss, Williamson, Fürstner, Goldscheider, P. Marie, Hess, and Bartsch. An argument which has been advanced in support of this hypothesis—namely, that a vessel with altered walls, and it may be even obliterated lumen, may be seen in the centre of the sclerotic patches—does not hold good in many cases, for patches may often be seen which are in no way related to vessels. Further than this, while vessel changes are met with in a considerable proportion of cases of disseminate sclerosis, there are cases of the disease on record, otherwise typical, in which the vessels have shown no morbid change. Moreover, when vessel changes are present the most pronounced alterations may be seen in parts of the central nervous system quite free from any other morbid change, while well-marked degenerated areas may be in no way related to vessels. There is much then to suggest that the vessels may be affected by, say, some toxin to which the other morbid changes met with in the disease are due; but that the vascular change can in no sense be regarded as the starting-point of all the anatomical changes, though of some of them it may be.

A further hypothesis to be considered is that of Strümpell, who, as a result of two observations in which he met with a combination of hydromyelia, central gliosis, and multiple sclerosis, suggests that possibly disseminate sclerosis is not a disease of exogenous origin at all, but one truly endogenous; and again, that it is a multiple gliosis depending on congenital influences, such as are operative in the case of multiple neuroma, fibroma, lipoma, and the like. In support of his contention he points to the fact that nearly all nervous diseases of exogenous origin are characterised by early affection of the nerve-cells and axis-cylinders, multiple sclerosis being the only exception. He further points out that if the disease begins in the glia, without primary affection of the neurons, it is easier to understand why the myelin sheath suffers alone, and why the axis-cylinder may be so long preserved. Then again, the fact that the disease usually manifests itself in young persons, and that its first manifestations may be traced even to childhood, is regarded as significant in this connection. Strümpell also points out that a disease may be thus endogenous, and yet the impetus to the disturbance may be the result of some exogenous influence; such as an acute disease or a trauma.

A careful review of the subject makes it clear that in the present state of our knowledge the origin of the disease cannot be definitely decided. The most prominent feature in the morbid picture appears in most cases to be the neuroglia proliferation; but whether this be primary, or secondary to a parenchymatous degeneration of the nerve elements, must necessarily be largely a matter of surmise until a greater number of cases have been studied histologically in the earliest stages of the disease. With regard to the vessels, however, the occasional changes in them can only be regarded, at most, as contingent elements in the disease.

Diagnosis.—Easy as is the diagnosis of disseminate sclerosis when its clinical manifestations are those which characterised the graphic picture of the disease drawn by Charcot in his classical account of it, nevertheless the diagnosis is a matter of extreme difficulty in many aberrant cases of the affection, a various group which, as we have already seen, is even larger than that of the so-called typical cases.

Hysteria.—Of all conditions of the nervous system none is more likely to be confounded with disseminate sclerosis than hysteria. Dr. Buzzard's statement—that disseminate sclerosis in its earlier stages is of all organic diseases that most commonly mistaken for hysteria—has every justification. The combination of symptoms unconnected with any one system and independent of anatomical distribution, in conjunction with the rapid variations and changes of the manifestations of the disease, make it easy for the two conditions to be confused. Both are met with most commonly in early adult life, and both may appear to depend on some mental shock, either alone or combined with some physical injury, perhaps of the most trivial character. Scanning speech and intention tremor may both be met with in hysteria; and it has been said that nystagmus also may occur in this affection. The latter statement is, however, probably erroneous; for in an extensive inquiry into the distinguishing features which characterise these two diseases, Souques never found this symptom present in hysteria. To a form of jerky unsteadiness of the globes, commonly associated with blinking of the lids which may occur in hysteria, I have already alluded.

Our difficulties in diagnosis are intensified, as we have seen, by the frequency with which hysterical manifestations supervene upon the organic basis; in such amount, it may be, as effectually to obscure the underlying structural defect and make errors in diagnosis of common occurrence.

On what points then are we to rely for a diagnosis between two conditions so easily confounded? Of primary importance, if present, is the detection on ophthalmoscopic examination of changes in the optic nerves; or, failing this, of a certain character of defect of vision. In both diseases disturbances of vision occur, but in hysteria no structural changes exist in the optic nerves. Next in order of importance is the detection of distinct nystagmus, which, as we have already seen, probably never occurs in hysteria; and, when present in disseminate sclerosis, is a symptom of great diagnostic value. Loss of control over the sphincters may occur early, and thus aid our diagnosis; though more commonly they are affected late in the course of the disease, after other symptoms characteristic of the organic affection have manifested themselves, and diagnosis is no longer in doubt. Another phenomenon which may occur early, and thus help us in diagnosing the organic affection, is the abolition of one or both knee-jerks,—a state of things which is, however, comparatively rare.

The well-known stigmata of hysteria, when present, though leaving no room for doubt that there is this functional element in the case, unhappily do not aid us in determining whether the functional manifestations have an organic substratum. Similarly, the influence of suggestion in modifying or clearing up the symptoms does not materially alter the position of doubt in which we may find ourselves.

Primary lateral sclerosis. — As the earliest manifestations of disseminate sclerosis may be a spastic paraplegia, without any evidence of affection of the superior extremities or territories presided over by the cranial nerves, it is obvious that in any case of the kind it may be quite impossible to say at the outset whether the paraplegia is the initial symptom of a disseminate disease; whether it is due to a primary sclerosis of the pyramidal tracts, or to some myelitic process limited to the spinal cord. It is only by carefully watching the progress of such cases that a correct opinion can be formed, and it is all-important in this connection to keep a careful watch on the optic nerves. Little less important is the detection of central scotoma, peripheral contraction of the visual fields, distinct nystagmus, or intention tremor; all of which naturally point to disseminate sclerosis. A word of warning, is, however, necessary in regard to nystagmus and intention tremor. I have had several opportunities of seeing cases of spastic paraplegia, presumably the result of myelitis limited to some part of the caudal half of the spinal cord, several years after they first came under my observation, in which slight nystagmoid movements, slight intention tremor, and increased activity of the arm-jerks were detected, symptoms which were not present when the patients were first seen. Opportunities of verifying the diagnosis in these cases have not occurred, but a careful examination of the clinical facts appear to me to justify the assumption that they are not instances of disseminate sclerosis. Why such late symptoms should arise in connection with a lesion of the caudal half of the cord cannot be discussed here; but such slight tendencies to nystagmoid movements and intention tremors occur in debilitated conditions; and in some instances, in which I have observed these late symptoms, the patients had been attacked by influenza after the occurrence of the paraplegia.

Ataxic paraplegia.—In this group may be included the combined degenerations of the spinal cord which occur in association with anæmia, marasmus, and the like; and much of what has been said in connection with spastic paraplegia is obviously no less applicable to these affections, in which also a provisional diagnosis is all that can be made in the early stages. In some of those exceptional cases of disseminate sclerosis, in which throughout the clinical course the manifestations are those of a spinal affection without any evidence of invasion of the brain, the symptoms may so closely resemble those of ataxic paraplegia as to make a diagnosis impossible. The tendency in some of these cases to improve for a time, and then to relapse, may increase the difficulty of diagnosis; but these ameliorations do not occur so frequently, nor are they, as a rule, nearly so great as in the case of disseminate sclerosis. An instance in which a peculiar form of paraplegia associated with anæmia, and supposed to be due to the cord changes met with in this affection, has recently come under my observation, where examination of the spinal cord revealed unmistakable evidence of disseminate sclerosis. In this case up to the end there was none of the characteristic symptoms of the disseminate disease to aid diagnosis.

Hereditary ataxia.—Inasmuch as we have seen that multiple sclerosis may occur in quite young children, it is sometimes difficult to distinguish between this condition and Friedreich's ataxy. A few instances have been recorded in which more than one member of the same family has been the subject of disseminate sclerosis; but this affection cannot be said to be a family disease, so that the occurrence of symptoms in more than one member of the same family is a diagnostic point of importance. Of further value in diagnosis is the absence of knee-jerks and the detection of any bodily deformity such as lateral curvature, pes cavus, and the like.

Even more difficult to discriminate may be the form of cerebellar heredo-ataxia, which begins between the ages of twenty and thirty years. In both affections there may be mental deficiency, optic atrophy, failure of pupil reaction, paralysis of ocular muscles, nystagmus, intention tremors of the superior extremities, a titubating gait, and increase of myotatic irritability. As in Friedreich's ataxy, chief reliance has to be placed on the discovery of evidence of affection of other members of the family as an aid to the diagnosis of the hereditary affection. It must not be supposed, however, that the occurrence of the affection only in one member of a family necessarily negatives the diagnosis of the hereditary affection, as other children may become affected subsequently. We ought especially to be on our guard if the eldest child of a family happen to be the one affected.

Pseudo-sclerosis.—Westphal described two cases in which there was the clinical picture of disseminate sclerosis during life, but no changes were found after death to account for the condition. Leyden and Langer have also met with this condition of things. The exact nature of these cases is doubtful, and they are far too rare to be of much importance. They may depend on some form of intoxication acting on the neurons with sufficient intensity to derange function without leading to structural change. On the other hand, it may be that with cruder methods of examination structural changes were overlooked which would have been detected by modern methods of examination. For this subject the reader is referred to a subsection on p. 92.

Tabes.—The class of case of this affection likely to be confounded with disseminate sclerosis is that in which motor weakness preponderates and ataxy is slight. Failure of the pupil to respond to light is rare in disseminate sclerosis, and, if present, would be probably associated with other evidences of implication of the brain by the disease; whereas in tabes it may be the only indication of the kind. Moreover, cases of disseminate sclerosis in which the knee-jerks are abolished are not common, and, when met with, muscular atrophy in the inferior extremities is not as a rule long delayed, as the sclerotic patch in the lumbar cord occasions destruction of the anterior horn cells. Besides this, the knee-jerks may be absent for a time and then return; while in tabes, after being really abolished, they do not return except where degeneration of the pyramidal tract has subsequently occurred, as in a case recorded by Drs. Hughlings Jackson and James Taylor.

Amyotrophic lateral sclerosis.—Whether the manifestations be limited to the spinal cord, or involve the bulb, the question of diagnosis from disseminate sclerosis in which atrophy of muscles is present may arise. The distinguishing feature is that in disseminate sclerosis paralysis precedes muscular atrophy, while in amyotrophic lateral sclerosis atrophy precedes paralysis, and the latter is proportional to the degree of atrophy.

Syringomyelia.—The class of case of disseminate sclerosis last considered may similarly suggest syringomyelia, the more so as nystagmus may exist in both diseases; but the trophic disturbances of the skin, and perhaps of the joints, preservation of tactile sensibility with abolition of the power of perception of painful and thermal impressions, together with the presence of lateral curvature, do not, as a rule, leave the diagnosis long in doubt.

General paralysis of the insane and disseminate sclerosis may resemble each other closely in some cases. Mental changes may occur in both; but dementia is present only in advanced stages of disseminate sclerosis, and is never so marked as in general paralysis; and though there may be a condition of bonhomie, there is never anything amounting to the grandiose state met with in a good many subjects of general paralysis. The resemblance of the speech defect is superficial, and on careful examination the differences are easy to distinguish. The paralytic speech is tremulous, but not scanning; and paraphasia literalis does not occur in disseminate sclerosis.

Although in general paralysis there may be intention tremor, which may be very difficult to distinguish from that of multiple sclerosis, as a rule there is no such difficulty, as it is more of a fibrillary tremor, affecting especially the facial muscles and tongue; and even when of greater range of oscillation the vibrations can be distinguished by their constant presence, irrespective of voluntary movement.

In spite of the attempt of Moncorvo and others to establish a connection between syphilis and disseminate sclerosis, an antecedent history of the former disease would lend weight to the opinion that the disorder of the nervous system was general paralysis rather than disseminate sclerosis.

Cerebro-spinal syphilis.—Cerebro-spinal syphilis may give rise to very similar symptoms; especially to optic nerve changes, spastic paralysis, and apoplectic attacks. But nystagmus, tremor, and scanning speech are wanting; while in disseminate sclerosis the sensory phenomena are in the background, and there are usually no meningitic symptoms: dementia is less common, as are also paralysis of cranial nerves and hemiplegia. In some cases it may be impossible to distinguish between the two diseases in any other way than by testing the effect of antisyphilitic treatment.

Cerebral tumour.—Mistakes in diagnosis may be made in the class of cases of multiple sclerosis in which hemiplegia occurs; it may be with headache, optic neuritis, and other symptoms of apparent cerebral origin.

Or the mistake may arise through ignorance of the fact that a tumour, especially if situated in the pons or crus, may give rise to an incoördination of movement closely resembling that seen in disseminate sclerosis, and occurring only on voluntary movement; and that nystagmus and incoördination may be indications of a tumour seated in the cerebellum. On the other hand, it must be borne in mind that a sclerotic patch involving the cerebellum, or one of its peduncles, may give rise to symptoms resembling those of tumour; although optic neuritis and pressure symptoms would of course be absent.

Although optic neuritis may occur rarely in disseminate sclerosis, it never assumes the form of choked disc; and there is an absence of evidence of increased intracranial pressure as evidenced by slow pulse, vomiting, and so on. Where it is not possible to arrive at a diagnosis, spinal puncture has been suggested as an aid, since increase of cerebro-spinal fluid would not be expected in disseminate sclerosis; but it is only where the symptoms suggest a tumour so situated as to be accessible to surgical interference that such a method of diagnosis seems justified.

Apoplexy.—As apoplectiform attacks sometimes occur in multiple sclerosis it becomes necessary to distinguish such attacks from the results of the rupture of a cerebral vessel or its occlusion. Such attacks in disseminate sclerosis are always transitory, and no evidences of descending degeneration appear. Further, the premonitory symptoms of true apoplexy are wanting, and there is an absence of the initial fall of temperature which accompanies the commencement of the attack.

Birth palsies.—Affections of this kind, in which the motor cortex of both cerebral hemispheres are involved, may be responsible for incoördination of movements of both arms and legs indistinguishable from the jerky movements of multiple sclerosis; furthermore there may be a closely similar defect of speech. The history as a rule suffices to distinguish these cases; but when it cannot be obtained satisfactorily some uncertainty may exist for a time. The progress of the two affections is so totally different that it cannot be necessary to extend the observations over any long period of time before arriving at a definite diagnosis.

Toxic tremors.—There is little likelihood of any real difficulty of diagnosis arising in this connection. In some cases of chronic alcoholism, in addition to tremor there may be nystagmus-like twitchings of the eyeballs. The tremor is fine and rapid, and affects the tongue as well as the hands; moreover, it may be aggravated by cutting off stimulants, and diminished by their administration.

Mercurial tremor persists apart from voluntary movement; and in cases where it occurs the patient is already in an advanced state of cachexia which cannot be mistaken. In addition to this, both in alcoholism and in mercurial poisoning, other symptoms which characterise the existence of disseminate sclerosis are absent.

Paralysis agitans.—So totally different are the pictures of the two diseases, that in spite of tremor being common to both, it is difficult to suppose that any real difficulty in diagnosis could arise; nevertheless most authors appear to consider it necessary to call attention to the points which distinguish the two diseases. Paralysis agitans occurs in much older subjects; the tremor is constant; and, even when extensive in range, is always regular. Added to this the mask-like face, fixed attitude and rigidity without increased activity of the tendon reflexes, make it impossible to confuse the two diseases.

Prognosis.—As soon as we have established our diagnosis a fatal prognosis becomes equally certain; but in few diseases is it more difficult to fix the probable duration of life with approximate certainty. We have noted the great tendency in this disease to apparent recovery, or considerable amelioration for long periods. It is obvious, therefore, how hazardous it would be to express too confident a prognosis when we are able to recognise the disease early. If the case is seen after one or two periods of apparent arrest or improvement, life is likely to be spared longer than in the case of a more or less steady progress without material remission. As a general rule life is not usually prolonged beyond two or three years after the clinical picture is sufficiently characteristic to leave no doubt as to diagnosis; but there are many notable exceptions to this rule; and, as already quoted, Charcot considered that patients in whom "spinal" symptoms only are present may live for twenty years or more. Symptoms indicating implication of structures in the medulla are of grave import, and may be taken to mean that a fatal issue may be expected much earlier than when such symptoms are not present.

So too the degree of care and attention that can be obtained in the way of good nursing materially affects the prognosis as regards the probable duration of life; more especially where there is a tendency to trophic disturbance of the skin, loss of control over the sphincters, or retention of urine requiring the use of the catheter.

Irrespective of the care that can be given in the nursing of the patient, the early occurrence of bladder troubles is an indication that the duration of life will be shorter than other manifestations of the disease would justify us in supposing; for in spite of all care that may be exercised in the nursing of such cases the risks of cystitis and secondary pyonephrosis are great.

Treatment.—The therapeutic prospects in disseminate sclerosis are gloomy in the extreme; no medicinal agent has the slightest effect in arresting the disease, or of retarding its progress. Various remedial agents have from time to time been vaunted, some no doubt in consequence of erroneous diagnosis in the first instance, and others in ignorance of the remissions which are common in the course of this mysterious disease.

The drugs to which we are justified in giving a fair trial are silver and arsenic. The former drug may be given with advantage, either as the nitrate or in the form of the chloride, in increasing doses by subcutaneous injection. Eulenburg has recommended that arsenic also should be similarly administered; but it is doubtful whether this mode of giving the latter drug has any advantages over its administration by the stomach.

According to Grasset, solanine diminishes the amount of tremor, but it has been suggested by Collins that where this drug has appeared to do good the diagnosis was at fault, the cases in reality having been hysteria.

No less disappointing are the results of hydropathic and electrical treatment, both of which have been recommended more especially by German authorities, but have failed to give satisfactory results in the hands of competent physicians. These measures should, however, have a fair trial in the earlier stages of this affection; and massage, with passive movements at the various joints, is of undoubted value in lessening the tendency to permanent contracture and to spasmodic rigidity.

But, although we can do so little to influence the morbid process directly, we can do much indirectly to retard the progress of the disease, and to make the time that remains to the patient a great deal more endurable than it would be without skilled medical aid and careful nursing. In the first place, it has been found that these patients, in the northern parts of the globe, are less comfortable in winter than in summer; so that, where it is possible, they should be sent to spend the winter months in warm climates. All depressing influences must be removed as far as possible; rest must be secured; and every means taken to maintain the general health at as high a standard as possible by open-air carriage or wheel-chair exercise, the administration of nutritious food, the exhibition of cod-liver oil, malt, medicinal tonics, and the like.

In the case of female patients pregnancy is especially to be avoided; for during the latter half of pregnancy, and it may be after delivery, the disease is more likely to advance rapidly.

Fatigue of all kind, both physical and mental, must be avoided; as must indulgence in wine and venery.

The occurrence of bedsores, cystitis, and similar complications call for the generally recognised means for their alleviation which will be found described in other parts of this work (*e.g.* vol. i. p. 432). Intercurrent affections must be treated on general lines if they occur in the course of the disease.

The time has not yet arrived when we can speak with any degree of certainty or confidence with regard to preventive treatment in this disease; but many facts in the etiology at any rate warrant our insisting on certain points in this respect, and in our expressing the hope that a not very distant future may bring us face to face with a rational and more hopeful means of dealing with so intractable a disease.

The infectious fevers appear to play so important a part in the causation of disseminate sclerosis, that it is incumbent on us to enjoin a sufficiently long period of rest and other means of recuperation after such illnesses; more especially when symptoms indicating derangement of the nervous system, no matter how slight in degree, have been observed during or after the acute disease.

Similarly, means should be adopted to reduce to a minimum the chances of intoxication in the various trades where metallic poisons are used.

All-important is it that we should warn those who have had initial manifestations of what may prove to be disseminate sclerosis, to take every care of themselves; to maintain their nutrition at a high standard; and especially to avoid depressing mental and physical influences, injury, exposure to wet and cold, and excesses of all kinds. I have said that women suffering from the disease should be especially warned against pregnancy.

PSEUDO-SCLEROSIS.—In 1883 Westphal published two cases in which the symptom group of disseminate sclerosis was present during life, but in which no corresponding changes were found after death. One of the cases was that of a young man, whose illness began at the age of eighteen years with weakness of the upper and lower extremities, and double vision; death resulted nine years later, before which time the following symptoms became manifest:—weakness of intellect; speech that was nasal without being quite scanning, and which finally became quite unintelligible; tremor of the head, lower jaw, muscles about the mouth and tongue; slowness of all movements; marked tremor of the arms on movement; and stiffness and uncertainty in the legs. The tendon jerks were active, sensibility was normal, as were the sphincters also; and the act of swallowing remained good up to the last. On necropsy, the brain, besides being anæmic, was very hard, especially in its posterior parts; otherwise nothing abnormal was found either on macroscopic or microscopic examination. No sclerotic patches and no tract affection.

The second case was also in a male subject. At the age of thirty years, soon after an attack of enteric fever, he became weak in his legs, and had a dead feeling in his hands. The progress was slow, but in time distinct physical disturbance became evident: speech became difficult and altered, often scanning; there was marked tremor of the arms and legs, with stiffness of the latter and a spastic gait. The tendon jerks were increased, and there was marked paradoxical contraction. Apoplectiform attacks with hemiplegic phenomena occurred, and in the course of the affection there were improvements and relapses. At times pain was complained of in the extremities, and there was slight disturbance of sensibility. There was no marked bladder trouble. Death occurred after an illness of ten years. Nothing was found at the necropsy to account for the clinical manifestations, and subsequent microscopic examination proved equally negative.

Westphal regarded as characteristic of the condition, for which he proposed the name "pseudo-sclerosis," the speech defect; the slowness of movements in the eye and face muscles, combined with a peculiar fixed expression; the physical disturbances; the apoplectiform attacks; the marked tremor in the extremities and the spastic phenomena, notably the increased tendon jerks; the paradoxical contraction; the motor weakness; the slightness of the disturbance of sensibility, and the normal state of the sphincters.

Strümpell has quite recently recorded two cases which closely resemble those published by Westphal. In the first of these, during the whole of the time that the patient was under observation, the diagnosis could never be quite certain. It seemed clear, however, that some severe organic affection must be present to account for the manifestations, the most likely condition appeared to be disseminate sclerosis, as there were marked scanning speech, great intention tremor of the arms, and spastic phenomena in the legs. This diagnosis seemed the more probable when, later in the course of the illness, apoplectiform and epileptiform attacks occurred. Yet Strümpell was never quite satisfied with the diagnosis of disseminate sclerosis, and considers that the following points militated against it:—the manifestations of the disease began as early as the age of twelve years, whereas, according to him, nearly all the cases of supposed disseminate sclerosis in children are open to question. The character of the disturbance of movement of the arms was not that usually seen with the intention tremor of disseminate sclerosis; and in the legs the spastic phenomena were not associated with ataxy, as is observed at any rate in most cases of disseminate sclerosis. On necropsy all that was found was slight degeneration of the crossed pyramidal tracts in the cervical region of the cord, and a small symmetrical patch of degeneration on the two sides in the upper cervical cord in the region of Gowers's tract, which areas of degeneration could be traced to the lower end of the medulla oblongata. Not a single sclerotic patch was found to support the diagnosis that seemed probable.

The clinical manifestations met with in Strümpell's second case also made it probable that disseminate sclerosis was the underlying pathological condition; but at the autopsy nothing abnormal was found, except that the white matter of the brain was abnormally firm, especially in the occipital region where it was quite leathery in consistence. On subsequent microscopic examination a very slight degree of degeneration of the crossed pyramidal tracts in the upper part of the cord was met with, as in his other case. Microscopic examination of the apparently sclerotic part of the brain proved that the nerve-fibres were intact, and that there was no increase of interstitial tissue.

Marie has regarded Westphal's cases as instances of hysteria; but in this opinion he receives no support either from Oppenheim or Strümpell, and the latter observer, who is, of course, quite familiar with those cases of hysteria which resemble disseminate sclerosis in their manifestations, could find nothing in the description of Westphal's cases, or in his own, to justify the assumption that hysteria was in any way concerned in the cases.

A consideration of Westphal's cases alone makes it seem possible that they depended on some form of intoxication acting on the nerves with sufficient intensity to derange function without leading to structural change; or that cruder methods of examination permitted structural changes to escape notice, which by modern methods of examination would have been detected.

The slight degenerative changes met with in the crossed pyramidal tracts in the cervical region of the spinal cord in Strümpell's cases certainly cannot be regarded as adequate to account for such severe disturbances of nerve function as were present in both cases during life.

Some important light has been thrown on the possible nature and etiology of this class of case by the findings in a third case which Strümpell has included in his paper. In this, as in the other two cases, the most probable diagnosis seemed to be disseminate sclerosis, while at the same time there was a great resemblance between the case and those of pseudo-sclerosis, both in regard to the clinical manifestations and the subsequent anatomical findings. There was a complete absence of any distinct sclerotic patches such as are found in disseminate sclerosis; instead of this the greater part of the brain was uniformly firm and leathery in consistence, just as was met with in more circumscribed area in two other cases of pseudo-sclerosis. No increase of interstitial tissue could be found to account for the increase of consistence. In the spinal cord slight but distinct degeneration of the crossed pyramidal tracts could be traced down to the lumbar region.

This brings the cases of pseudo-sclerosis more into line with the diffuse cerebral sclerosis of which in children, according to the investigations of Ganghofner, hereditary syphilis is probably a cause. Strümpell thinks that this is very likely, and sees no reason why acquired syphilis should not be operative in the cases of diffuse cerebral sclerosis that occur later in life. This would explain why so many of the manifestations of the condition resemble those of general paralysis of the insane. Moreover, according to Strümpell, it is possible that other noxious influences, such as alcohol for instance, may be equally effective.

Important as are these cases of pseudo-sclerosis, they have not been met with often enough to form a serious obstacle in the diagnosis of disseminate sclerosis.

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TABES DORSALIS

SYNONYMS.—*Locomotor ataxy, Posterior sclerosis of the cord.*

Historical.—Early in the present century certain German physicians had constructed a rough account of this disease, and Romberg in his *Text-book of Nervous Diseases* (1846) gives an authoritative description of it. In this country observations had been made by Stanley, Todd, and Gull, and in France by Ollivier and Cruveilhier; but out of Germany the disease had not been recognised as a uniform group of symptoms. Hence, when Duchenne's masterly clinical sketch appeared (1858) it was generally accepted as the description of a new disease. Duchenne, emphasising a fact on which Romberg had laid little stress, showed that, while muscular power may be retained, the orderly performance of movements is increasingly impaired, and he therefore proposed the well-known name, "progressive locomotor ataxy." His writings, backed by the authority of Trousseau, obtained for the disease a wide recognition, and both clinical and pathological records became more general. Thus important observations were made by Charcot, Vulpian, Bouchard, and others; monographs upon it appeared also, such as that by Topinard in France and Leyden in Germany. Among English physicians Dr. Hughlings Jackson wrote particularly on the optic atrophy, and Lockhart Clarke on the morbid anatomy. On this latter subject Pierret began a series of papers in 1870, pointing out a special part of the posterior columns as the starting-point of the spinal lesion. A few years later came Charcot's famous lectures, which awoke much interest both in this disease and in the subject of nervous diseases generally. In these he dwelt particularly on certain less known symptoms, such as the joint disease (which had been previously described by him), the visceral crises, and so on. Accounts of the joint symptoms were also published by Clifford Allbutt (1869) and by Buzzard. In 1876-78 Erb published his well-known article in von Ziemssen's *System of Medicine*, ably setting forth the whole subject of tabes.

The phenomena of tendon reflexes, simultaneously broached by Westphal and by Erb in 1875, gave a fresh impulse to the study of this disease; since by Westphal's sign (loss of knee-jerk), and by Argyll-Robertson's sign (loss of the pupillary reflex under the stimulus of light), we are enabled to diagnose its early stages with far greater precision. From that time an ever-growing number of observations has appeared, among which I may specially mention divers interesting clinical histories by Buzzard; statistics on the relations of syphilis to tabes by Fournier, Erb, and others; observations on peripheral nerve degeneration in true tabes by Pitres and Vaillard, and by Oppenheim; the simulation of tabes by peripheral neuritis (pseudo-tabes) by Déjerine; the article upon Tabes in the *Text-book of Nervous Diseases* by Gowers, and the lectures upon this subject by Marie.

Etiology.—The etiology of tabes is not fully understood; possibly it is complex, and not always uniform. Statistics by most competent observers show that *syphilis* is an antecedent so common that we can hardly avoid including it in the causation. But the history of such syphilis is usually remote, and often it has been seemingly slight—a chancre without constitutional symptoms; and as tabes, generally speaking, is not amenable to anti-syphilitic treatment, it may be asked, not unnaturally, what possible connection there can be between the two diseases. No one, certainly, would allege that tabes is pathologically identical with syphilis—that it is a gummatous disease of the nerve-centres such as could be removed by mercury or iodide; it is alleged rather that syphilis in some way lays the foundation for it. If we ask in what way, some authors urge the analogy of post-febrile and toxic paralysis. As diphtheria causes a poison to be elaborated which, acting on the nervous system, produces post-diphtheritic paralysis, and as certain poisons, such as ergot or lathyrus, have a selective action on various strands of the spinal cord [vol. ii. p. 799 and p. 805], so (it is thought) may the nervous system and the posterior columns in particular be poisoned and degenerate as the result of bygone syphilis. Other authors maintain that the degeneration is secondary to some well-recognised syphilitic lesion, such as an arteritis, or to a spinal meningitis. However this may be, the statistical facts remain that most, though not all, of the patients who have tabes have also had syphilis.

Another great factor in the etiology is *sex*. Men suffer in a far larger proportion than women. The reason of this fact we do not know, nor how to reconcile it with a purely syphilitic etiology. As to *age*, the disease commonly manifests itself first in the fourth or fifth decade of life, though there may be exceptions to this, and a close inquiry into early symptoms may carry its origin farther back than was suspected. In children true tabes is almost unknown. The few recorded cases (about ten in number after excluding cases of Friedreich's disease) do not quite tally with the typical disease as seen in adults. As to *heredity*, a "general nervous heredity" is often recorded; that is to say, nervous diseases of various kinds may figure largely in the patient's family history; but the disease itself is not transmissible, unless it be in certain very rare instances, nor is it in any sense a "family" disease. Some curious instances may be here noticed, in which a husband and wife have suffered from tabes. The disease is sufficiently uncommon in women to make it unlikely that this was a pure coincidence, and there has been in such cases a history of syphilis affecting the pair, which in all probability gives the clue to the origin of the tabes.

Turning from general and remoter influences to proximate and determining causes, we find the following enumerated: exposure to cold; over-exertion, especially when combined (as in military campaigns) with exposure and privation; sexual excess; traumatism. Sometimes the history reveals none of these things, sometimes one of them assumes such prominence as to convince the patient at any rate of its importance. Their proper estimation is difficult, and necessitates careful inquiry into the circumstances of each case, for early symptoms, forgotten or disregarded, may have preceded the action of the supposed cause, which may have merely aggravated but not initiated the disease. This remark, I think, applies particularly to histories of *injury* causing either a general shock or a shock to the spine; such as blows, falls, railway collisions, and the like. A patient may become ataxic after such an injury, but we must not assume that his disease originated in that way till we have learned that there is no previous history of lightning pains, squint, or other early symptom of tabes. But there may be another sequence of events. An injury to a limb is followed by lightning pains, which begin at the seat of injury and subsequently become generalised; the other phenomena of tabes appearing in due course. The relation of cause and effect seems at first sight clear; and the explanation generally given is that a neuritis was set up which spread upwards by the posterior roots into the cord. However, the possibility of such an extension seems rather doubtful; and Hitzig, from an analysis of such cases, concludes that there are very few which, even from the clinical side, are above criticism. On *sexual excess* a traditional stress has been laid; among modern and independent writers Erb ranks it as a factor in the etiology of tabes. It is obviously a difficult subject for inquiry, and the more so as abnormal sexual excitement has been reckoned among the early symptoms of the disease.

I incline to the view that there is in all cases some remoter influence, which is generally past syphilis, or in some few cases other factors as yet unknown; and that upon this may follow the nerve degeneration, either without definite proximate cause, or determined by some of the proximate causes we have just enumerated.

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Pathology.—Tabes is a primary progressive degeneration of the first afferent (sensory) projection systems of neurons, by which peripheral sensations are cut off from various parts of the central nervous system; the commonest and most obvious anatomical change being degeneration of the posterior spinal roots and the posterior columns of the spinal cord. The clinical phenomena characteristic of this disease depend upon the systems of neurons which are undergoing degeneration, and on the extent as well as on the rapidity of the process. Stanley, Steinthal, a pupil of Romberg, and Cruveilhier had all described sclerosis of the posterior columns of the cord in cases of so-called paraplegia: but it was Todd (1847) who first discovered the pathology of tabes; for he correctly explained the paralysis of the lower extremities, and associated it with the loss of the guiding sensation coming from the lower extremities. In the *Cyclopædia* *of Anatomy and Physiology* Todd explained thus the incoördination of movement by the sclerosis of the posterior columns, fully justifying Sir William Gowers in claiming for him the discovery of this disease.

> "Two kinds of paralysis of motion may be noticed in the lower extremities, the one consisting simply in the impairment or loss of the voluntary motion, the other distinguished by a diminution or total loss of the power of co-ordinating movements. In the latter form, *while considerable voluntary power remains*, the patient finds great difficulty in walking, and his gait is so tottering and uncertain that his centre of gravity is easily displaced." He states the cases are chronic, and in two examples of this variety of paralysis (sensory) he predicted disease of the posterior columns, and such was found to exist on post-mortem examination."

Turck and Lockhart Clarke were the first to investigate the disease in the spinal cord microscopically, and to describe the wasting of the fibres.

Morbid anatomy.—After opening the spinal canal and slitting up the dura mater, it will be observed that the pia arachnoid is thickened over the posterior surface of the cord, which is flattened, and presents a grayish or grayish red aspect; moreover, the posterior roots are thin, flattened, and atrophied, although the degree of wasting is not necessarily uniform; they also present a gray appearance like the posterior surface of the cord. On removing the cord, and cutting it transversely, the degeneration is found to be limited to the posterior columns, which are considerably shrunken, and of a grayish or grayish red colour, contrasting strongly with the white antero-lateral columns. This degeneration is usually much more obvious and advanced in the posterior columns of the lumbo-sacral region; likewise the posterior roots entering into the formation of the cauda equina are, as a rule, atrophied to a greater degree than elsewhere. Normally the posterior roots are two or three times as large as the anterior; but in advanced cases of tabes they may, in the process of degeneration, waste to such a degree as even to be smaller.

The degenerative process is not limited, however, to the afferent spinal projection systems; various cranial nerves may be atrophied, and the peripheral nerves in many cases may exhibit degenerative changes. The gray atrophy of the optic nerve is obvious to the naked eye; but microscopical examination, aided by the various selective methods of staining, has enabled us to associate most of the symptoms of tabes with definite anatomical changes in the peripheral and central nervous systems. The Weigert, Weigert-Pal, Marchi and Marchi-Pal methods show that the degeneration of the posterior columns of the spinal cord is a system degeneration of exogenous origin precisely similar in anatomical distribution to that produced by section of the posterior roots; or, in the case of the lumbo-sacral region, to that produced by a tumour of the roots of the cauda equina. Flechsig, by the embryological method, demonstrated that the fibres of the posterior columns are derived from two sources: (i.) exogenous central projections of the T-shaped processes of the nerve-cells of the posterior spinal ganglia; (ii.) endogenous projections from cells of the gray matter of the cord: the former are degenerated in tabes, the latter are not; and this is the reason why, in the lower lumbar region of the cord, a small oval area of undegenerated fibres may be seen, even in advanced tabes, occupying the median portion of the posterior column. This undegenerated area consists of fibres derived from cells of the gray matter of the cord, and is named the oval area of Flechsig; it develops at a different period of time from the exogenous fibres. Leyden was the first to point out that tabes is a systemic degeneration and an extension of the lesion of the roots. Now, it is impossible to conceive that vascular changes, or impaired nutrition by an insufficient supply of blood, could produce in such a small area as the posterior columns of the spinal cord a degeneration of the fibres of exogenous origin, sparing those of endogenous origin and the adjacent fibres of the lateral column. Neither can we believe that the overgrowth of neuroglia tissue at the expense of the noble elements is anything more than secondary, and proportional to the parenchymatous degeneration. The old view was that it is a primary sclerosis of the posterior columns of the spinal cord, by which the nerve-fibres are gradually strangled. If this be so, why does not the neuroglia interfere with the nutrition of the endogenous fibres? Has it a selective action for one system? It is much easier to assume that it is a trophic change in the posterior spinal ganglion neurons. These are really complex cells with T-shaped processes: one branch of the T becomes a peripheral sensory nerve that terminates in a sensory end-organ or arborisation; the other passes by the posterior root to the cord, when it may immediately break up into a terminal arborisation in the gray matter; or it may run a variable distance in the posterior column before breaking up into its terminal arborisation (*vide* Fig. 1).

The dictum of Virchow, "A cell nourishes itself and is not nourished," may be applied to the pathology of tabes. A poison long present in the system can so lower the vitality of the cells of the body as to induce premature decay. It was shown first by Fournier, and subsequently by Erb, Gowers and many other eminent authors, that in a majority of the cases of tabes there is a history of syphilis. The clinical phenomena depend, as I have already remarked, upon the system of neurons affected, and the extent and rapidity of the process. There can be no doubt that, pathologically speaking, tabes and general paralysis are of the same nature; namely, a parenchymatous degeneration with secondary overgrowth of neuroglia due to atrophy of the noble elements. I have met with cases which began with tabetic symptoms and ended in general paralysis; general paralysis of the tabetic type is a far less rare condition. In the former the afferent projection system of neurons was affected primarily, and the neurons of association later; whilst in the latter the two were affected simultaneously. The symptoms entirely depend upon the anatomical distribution of the degenerative process. The thickening of the pia arachnoid, the hyaline thickening of the vessel walls, and the overgrowth of glia tissue met with in both diseases, may be explained by the irritation of the products of degeneration, inducing formative proliferation of the neuroglia elements, which receive an increased supply of nutriment, as the blood-supply can no longer be utilised by the dying nervous elements. It must be remembered, moreover, that the nerve-cell is a perpetual element—it is incapable of regeneration; unlike the cell of a gland, its restoration is impossible. Consequently, a poison like syphilis may lower the vitality and diminish the durability of certain systems of neurons, and given stress, injury, cold,

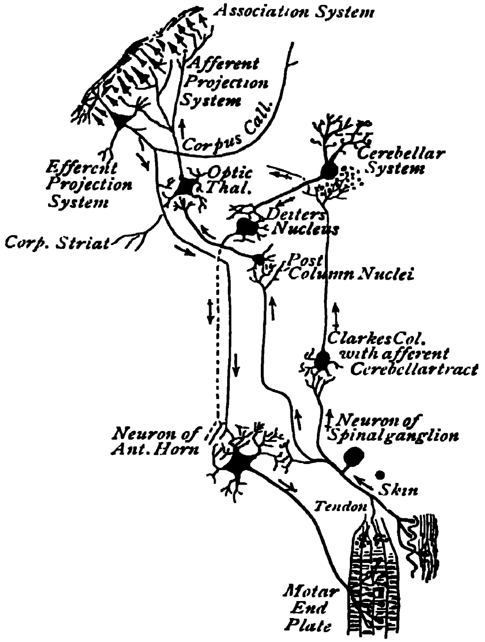

FIG. 1.—Diagram illustrating the afferent, efferent, and association systems of neurons. It will be observed that there are three nervous circles. In voluntary movements impulses are travelling along all these systems. In tabes there is degeneration and interruption of the afferent path in spinal, cerebellar, and cerebral systems; consequently incoordination of voluntary movements. Diagrammatically the posterior spinal ganglion is represented as giving off three processes; more probably it only gives off one.

or sexual excesses as immediate factors, the balance of repair and waste is overturned, and the neurons die gradually and prematurely. In *ergotism* and *pellagra* very similar systemic degenerations have been described (vol. ii. pp. 793 and 800); but, as neither experimental anæmia nor even starvation produces any appreciable wasting of the nervous system, it is probable that systemic degenerations are due to a progressive defective metabolism in the neuron itself. The body of the nerve-cell is the principal trophic centre of the neuron; the more remote parts of the neuron will therefore suffer first, namely, the collaterals and terminal arborisations, and this decay gradually spreads back towards the body of the cell, causing atrophy of the fibres in the posterior columns and posterior roots. The peripheral portion of the T-shaped process also may undergo degeneration (Westphal, Déjerine, Pitres and Vaillard). Oppenheim and Siemerling have described degeneration of the peripheral nerves, especially of the cutaneous sensory nerves.

It is very difficult to examine the end-organs, but Batten and other observers have described changes in the muscle spindles. Dr. Goldscheider states that the degeneration of the nerves is more marked at the periphery, and diminishes as the ganglion is approached. Still it is undoubted that the most marked change is in the central process of the neuron, and degeneration of the peripheral nerves is not discoverable in every case. It may therefore be asked why, if it is a trophic disturbance of the neuron, should the degenerative process affect its central projection principally, and in some cases only? Why should so many careful observers fail to find any noteworthy changes in the cells of the posterior spinal ganglion? I have found that examination of the spinal ganglion by Nissl's method after section of the posterior roots failed to show any changes in the cells, whereas section of the mixed nerve produces chromolytic changes in the cells of the posterior spinal ganglion as well as those of the anterior horn. Van Gehuchten asserts that after section of the mixed nerve the spinal ganglion cells undergo a degenerative atrophy, whereas the anterior horn cells recover completely in time. These facts appear to me of some importance in explaining those before mentioned relative to the morbid change in the spinal neuron in tabes; and also the probability that the peripheral portion of the T-shaped process represents the axon. In section of the posterior roots the ganglion cells are not cut off from peripheral impulses; in section of the mixed nerve they are. It is possible, therefore, to suppose that while the peripheral sensory nerves are intact (and we have seen that they degenerate at a later period and to a less degree than the posterior roots), the ganglion of which they are projections will not undergo atrophic changes. It may also be that the peripheral end-organs of nerves exercise a trophic influence; for it is asserted by Mackenzie that regeneration of a divided nerve may take place from the periphery.

We shall now consider in detail the changes which have been found in the nervous system by microscopical examination. The changes in the spinal cord are due to degeneration of the posterior root fibres. If a posterior root is cut—for example, the first sacral in the monkey—and the cord be examined at different levels, it will be found that there are four sets of fibres of different lengths; namely, two sets of fibres which almost immediately end as a brushwork of fibrils in the gray matter:—these are (i.) very fine fibres which form the cap to the posterior horn called Lissauer's tract; (ii.) another set of short, coarse fibres which pass forwards to the middle of the posterior horn and then run into the gray matter, terminating in an arborisation around the cells of the anterior horn. These two short sets of fibres are concerned with spinal conduction: the fine fibres of Lissauer's tract, from analogy of position with the so-called ascending root of the fifth, are possibly purely sensory; the coarse fibres are connected with the spinal reflex arc, and are instrumental in the maintenance of muscular tonus and the spinal reflexes. At first all the degenerated fibres are in the postero-external column, but as each successive healthy root comes in, the degenerated fibres which have not entered the posterior horn are pushed forwards and towards the middle line; as soon, however, as the posterior vesicular column of Clarke begins to appear at the level of the first or second lumbar, a number of degenerated fibres run forwards, terminating around the cells of this column. These fibres, therefore, carry impulses to the cerebellum; and since the entry of the first sacral root is some distance from the first lumbar segment, it proves the probability that every spinal root contains fibres which transmit impulses to the cerebellum (Fig. 1). These cerebellar afferent fibres are of medium length, and as soon as they have disappeared only one set of fibres is left; these take a direction backwards and upwards, forming a triangular area at the hinder part of the posterior median fissure—the column of Goll: this tract can be traced up to the nucleus of the funiculus gracilis. Goll's column forms the first segment of the direct sensory path to the opposite cerebral cortex. I have found, in studying the degeneration resulting from section of the first sacral posterior root, that this is by far the most important root entering into the formation of the column of Goll; and this fact is of interest when taken in conjunction with another, namely, that it is the root which especially supplies the sole of the foot. Section of roots in the upper lumbar and dorsal regions produces very trifling degeneration in Goll's column; most of the root fibres from these regions convey, therefore, either spinal or cerebellar impulses, not cortico-cerebral. Marie distinguishes three varieties of tabes according to the anatomical lesion: (i.) ordinary tabes; (ii.) superior or cervical, in which the knee-jerks may remain intact; (iii.) cerebro-bulbar; but hardly any two cases are precisely alike in their symptoms or in the anatomical distribution of the degenerative process. Certain tracts in the posterior column degenerate earlier than others. Charcot and Pierret showed that in the external third of Burdach's column there is very early an area of degeneration which they termed the bandelette. Marie states that in early cases and in certain varieties of tabes the degeneration in Goll's column may be little marked. Other cases, on the contrary, show extreme degeneration. It has been already pointed out that the fibres entering Goll's column come especially from the fifth lumbar and first and second sacral roots, so that if these roots were not much affected there would not be much sclerosis in Goll's column.

The zones of Lissauer—the fine fibres which form a cap to the extremity of the posterior horn, extending a short distance along the external and internal borders of it—degenerate and disappear in the early stages of tabes (*vide* Figs. 2, 3, 4). It is the lesion of the external portion of these zones which has been incorrectly described by certain authors as sclerosis of the lateral column. Another situation in which early degeneration is said to occur is the terminal arborisation of the root fibres around the cells of Clark's column (Fig. 7). Certain groups of fibres enjoy a particular immunity, and can be seen intact when all the rest of the posterior column is sclerosed: (*a*) the median oval area of Flechsig; (*b*) cornu commissural bundle-zone of Westphal (*vide* Fig. 2); (*c*) the postero-external angle of the posterior column; but this group does not offer the same resistance as the other two, which are certainly of endogenous origin.

The changes in the cord are usually more or less symmetrical; but the posterior roots are not always equally affected, and, of course, the site and extent of the spinal degeneration will vary accordingly.

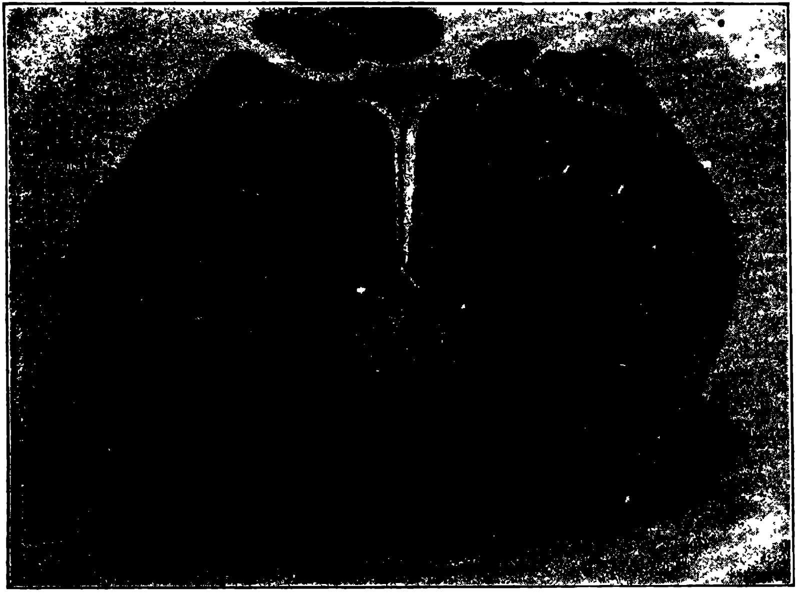

Fig. 2.—Photomicrograph of a section of the spinal cord at the level of the 5th lumbar segment, from a case of advanced tabes. There is a complete degeneration of the exogenous fibres of the posterior columns. In the middle line two oval areas can be seen which still contain undegenerated fibres of endogenous origin. The root fibres entering the posterior horn are entirely destroyed, also the posterior roots are completely sclerosed.

As a rule the lumbar roots are first affected; but in rare cases the mischief may begin in the cervical region (Figs. 4 and 5), and leave the legs intact. I have such a case of cervical tabes under my care at present. Sometimes, but rarely, cervical and dorsal regions may be affected simultaneously. In the brain the cerebral nerves and their ganglia may be degenerated. It is seldom that the olfactory nerve is affected; Pierret has described atrophy of the nerve in a case of tabes in which there had been anosmia. Visual defects and blindness are not at all uncommon, and these are due to a gray degeneration of the optic nerve. The ganglion cell layer of the retina corresponds to the nerve-cells of the spinal ganglia; first the nerve-fibres of the optic nerve are affected, and sooner or later the cells of the retina also (Möbius). The process also spreads centralwards towards the corpora quadrigemina and internal geniculate bodies. The permanent ocular palsies are occasioned by a degeneration of the oculo-motor nerve nuclei. The fine fibres between the cells in the nuclei are atrophied; the cells themselves are shrunken and pigmented and partly destroyed. With the degeneration of the nuclei we find degeneration of the root fibres and of the peripheral nerves of the eye muscles. In some cases these are affected alone, the nuclei being unchanged.

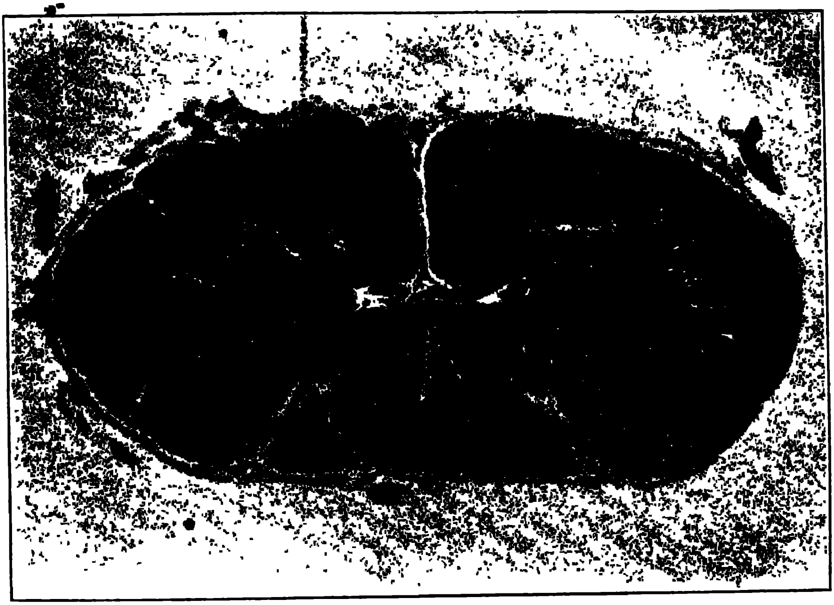

FIG. 3.—Photomicrograph of a section of the spinal cord at the level of the 8th cervical from a case of tabetic general paralysis. There is advanced sclerosis of the posterior columns. The cornu commissural fibres of Westphal, endogenous in origin, are seen to be undegenerated.

The ascending root of the fifth, the fibres of which spring from the Gasserian ganglion, has been found sclerosed in a case in which there were symptoms during life pointing to affection of the fifth (Oppenheim). Likewise the solitary bundle which consists of afferent vagus and glossopharyngeal fibres was found sclerosed. I have found partial sclerosis of this bundle in a case of cerebro-bulbar tabes in which during life there was loss of taste. Deafness observed during life has been found after death to be due to atrophy of the auditory nerve. Besides the oculomotor nuclei, another purely motor group of nerve-cells may be atrophied, namely, the hypoglossal; and hemiatrophia linguæ has been found as a result of atrophy of the hypoglossal nucleus of the same side. Slight changes in the cerebral cortex have been described, which correspond to those of general paralysis, but are less in degree. I have seen several such cases in which the dementia had been only slight, and the tabetic symptoms very pronounced. Changes in the peripheral nerves are very often present in the cutaneous nerves, and in long-standing cases a portion of the muscular nerves are sclerosed. In bone disease the nerves supplying the bone have been found degenerated; and in the neighbourhood of a perforating ulcer and such like trophic disturbances extensive degeneration of the nerves has been observed. The degeneration of the nerves is more marked at the periphery, and the nearer, centrally, the nerve is examined the fewer degenerated fibres are there found. Although degenerated fibres exist in the nerves entering muscle, yet the anterior roots are normal; this fact, however, can be accounted for, as Prof. Sherrington has shown that after section of all anterior spinal roots entering into the supply of a muscle a number of fibres proceeding to the muscle still remained normal; these are connected with the muscle spindles, and are the afferent sensory fibres of muscle. It is, therefore, in all probability these fibres which are degenerated. This does not, however, explain the degenerated fibres in the oculo-motor nerve, because he has shown that these contain no muscle spindles, and they degenerate completely when the nerve to the muscle is divided.

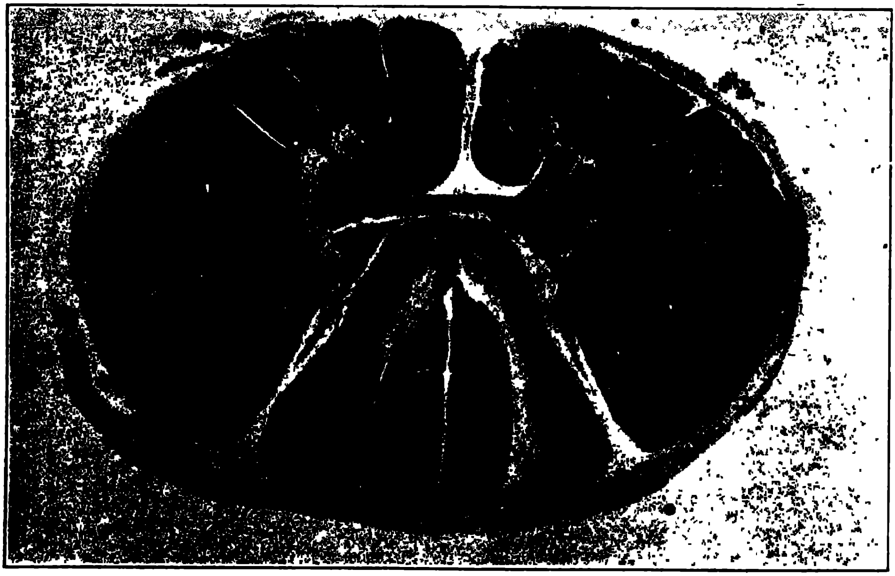

FIG. 4.—Cervical tabes after Marinesco. Section at the level of the 3rd cervical root. The lesion is asymmetrical, whilst on the left there is only ascending tabetic degeneration; on the right there is also local degeneration. The middle radicular zone contains fewer fibres than on the opposite side. The posterior median columns are intact, as they consist of the healthy sacro-lumbar and dorsal root fibres which enter into the formation of Goll's column.

A consideration of the anatomical distribution of the degeneration shows that it is especially an affection of the afferent projection system by which the central nervous system, spinal cord, cerebrum, and cerebellum, are gradually and progressively deprived of normal peripheral stimuli.

The nature of the degeneration.—Microscopical examination of the spinal cord shows the myelin sheath of the nerve-fibres diminished or destroyed; the axis-cylinder process may be swollen in one place, attenuated in another, and generally irregular in thickness or completely atrophied; the neuroglia is increased at the expense of the parenchyma, and a large number of Deiter's cells are visible. Nearly the whole of the posterior columns in the lumbo-sacral region may be destroyed, leaving only the cornu commissural and oval areas of endogenous fibres. The vessels are often thickened in the sclerosed area, and not elsewhere; this change is secondary to the degeneration, not causal. The walls of the arteries are often thickened, and there is hyaline degeneration of the media; sometimes the vessels are so much thickened by this degenerative process as to become almost obliterated, especially when the sclerosis is advanced. The pia arachnoid membrane is also thickened, and often presents the appearances of chronic inflammation. That tabes is a widespread process of degeneration, primary in origin, and not secondary to vascular change, is shown by the fact that the vessels of the retina are unaltered, even in advanced gray atrophy.

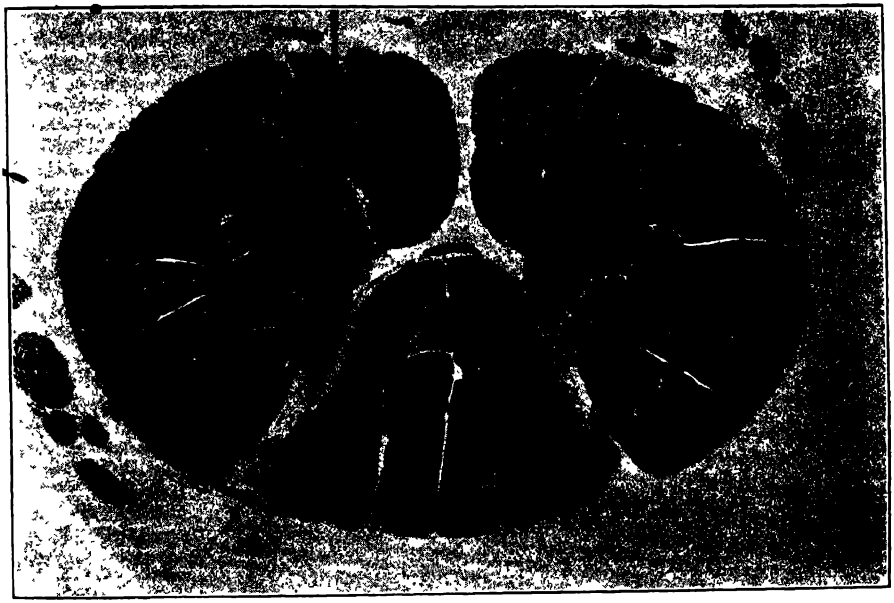

FIG. 5.—Cervical tabes after Marinesco. Section at the level of the 5th cervical. The same asymmetry of degeneration is observed in the posterior external columns, and Goll's column is intact.

Atrophy and degenerative changes in the cells of the spinal ganglion have been described by Stroebe, Wollenberg, and others. I have myself examined—by Nissl, Stroebe, and Pal methods—four cases of tabes, two of which were general paralytics, in which there was very advanced posterior root degeneration (Fig. 6), as well as almost complete sclerosis of the posterior columns (Fig. 2). In two cases all I could find were increase of pigment in the ganglion cells, nuclear proliferation in the capsule and interstitial tissue, and sometimes vascular congestion; generally speaking, there was inflammation of the loose connective tissue surrounding the ganglion. In another case, one of advanced tabes, I found, by Nissl's method, as well as the changes above mentioned, some apparent atrophy of the cells in the 4th and 5th lumbar spinal ganglia. Some of the sections showed a few groups of cells in a state of advanced degeneration; the capsule either empty or containing only a little mass of pigment. In a case of sclerosis of the posterior columns, combined with lateral sclerosis in a syphilitic patient, a number of the cells stained intensely purple by Pal and red by Stroebe method (*vide* Plate). These cells had undergone fatty degeneration, and the nucleus was no longer visible. I have seen similar appearances in the ganglia of a case of tabes, but not so marked. It is therefore probable that the ganglion cell is the last portion of the neuron to die, and that the process begins in the terminal twigs and spreads backwards to the body of the cell, but with greater rapidity in the central projection than the peripheral. Babes and Kremnitzer have described an atrophy of the plexus of fine fibrils which form a network around the capsule. These fibres, they state, come from cells in the spinal cord. Dehler has shown the existence of cells with a T-shaped process in the sympathetic ganglia: these may form the white ramus which connects the posterior spinal ganglion with the sympathetic ganglion; but I am not aware that any definite changes have been described in the sympathetic, or in this ramus; however, degeneration of the sympathetic is extremely difficult to recognise by any methods at present in use.

Can we apply our knowledge of the before-mentioned systemic degenerations in the spinal cord to the explanation of some of the important clinical phenomena of tabes? Reference to the diagram (Fig. 1) will help to explain some of the phenomena of tabes; namely, the diminution of tonus in the muscles, the incoördination, the absence of the knee-jerk, the ataxic gait, Romberg's symptom, and the various disturbances of sensation.

The afferent system of the neurons of the spinal ganglion convey sensations from the skin, the muscles, and the tendons and joints, and these sensations travel by three sets of fibres: (i.) *Short*, forming the spinal reflex arc; (ii.) *Medium* length fibres which break up into a brushwork around the cells of Clarke's column, the axis-cylinder processes of which form the direct cerebellar tract; (iii.) *Long fibres*, which form Goll's column and break up into a terminal arborisation around the cells of the nucleus gracilis. These fibres convey kinæsthetic impulses to the sensori-motor region of the cortex by the fillet and the thalamus opticus, and are brought

[redacted]

into relation with the efferent pyramidal system. There are thus *three nervous circles*—spinal reflex, cerebellar, and cerebral. The true motor neuron which controls the muscle is situated in the anterior horn; we know that in tabes this is unaffected—therefore the muscle does not waste; nor is there, except in the late stages, any paralysis or loss of strength of voluntary movement, only incoördination. Jendrassik, Fränkel, and others have shown that there is a marked loss of tonus. Whence comes this loss of tonus? It is due, like the loss of myotatic irritability and the consequent absence of the knee-jerk, to the break in the reflex spinal arc occasioned by the degeneration of the spinal roots, and of those fibres which run forward through the root and the base of the posterior horn, terminating in an arborisation around the anterior horn cells. By this degeneration the motor neurons in tabes are deprived of the normal stimuli which serve to maintain the reflex tonus and myotatic irritability (*vide* article "Tendon Reflexes," vol. vi. p. 529).

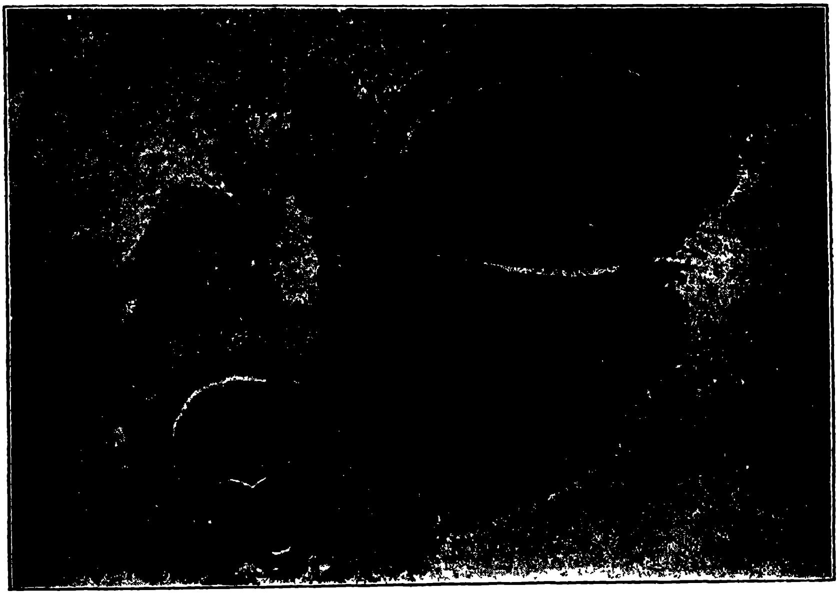

Fig. 6.—Photomicrograph of a transverse section of the posterior root (3rd lumbar) from a case of advanced tabes. The small dark round bundle of fibres is the undegenerated anterior root, the large sclerosed root is the posterior. Section stained by Weigert method. Magnification, 20 diameters.

The influence that sensations coming from the periphery have in maintaining muscular tonus was strikingly shown by the experiments which Dr. Sherrington and I made on monkeys. We found that immediately after dividing the posterior roots supplying the limb there occurred a great diminution in tonus in the muscles of the limbs; we also observed in two animals shortly after the operation, and for some considerable time afterwards, a marked incoördination closely resembling tabetic incoördination in man. If the limb was rendered quite "apæsthete" by dividing all the sensory roots supplying it, the animal was unable to use its hand for volitional movements; *there was a sensory paralysis*, although associated movements at the elbow and shoulder were frequently observed. This loss of volitional power over the hand was not due to injury of the efferent tract, as was proved by the fact that stimulation of the cortex evoked all the movements even more readily than on the sound side. The movements obtained by stimulation seemed to me in some instances to have a greater excursion than normal. This, I believe, is due to the loss of tonus in the muscles opposing the action; for example, extension of the wrist seemed to be greater and more sudden than normal owing to the loss of tonus in the flexors. By reference to Fig. 1 it will be observed that the limb of such an animal is practically cut off from the central nervous system. Except by vision the animal is unconscious of the existence of its limb, and without a knowledge of the sense of position it is unable to ideate a movement or to appreciate the successive changes which occur in the parts moved; consequently there is a paralysis of voluntary movement. It is only in advanced stages of tabes that there is actual paralysis; and an examination of the spinal cord in the paralytic stage shows complete destruction of the exogenous fibres of the posterior columns.

The experiments of Vierordt and Heyd show the importance of the cutaneous sensations coming from the soles of the feet; these observers found that by anæsthetising the soles of the feet instability and oscillation occur on closing the eyes. Tabetics, who did not show Romberg's symptom, exhibited it if the anæsthesia of the soles of the feet had been produced artificially. Clifford Allbutt, Leyden, Charcot, Vulpian, and others have explained Romberg's symptom by the falling away of cutaneous sensibility in the soles of the feet. There is no doubt that this increases the instability, but many tabetic patients exhibit this phenomenon when the ordinary methods of testing show perfect cutaneous sensibility in the feet. It is a curious fact that sometimes an ataxic patient can stand with opaque glass in front of his eyes, whereas he will immediately sway to and fro on closing his eyes. I have seen several cases of tabes, beginning with optic atrophy and ending in complete blindness, remain for a long time in the pre-ataxic stage, although they had lightning pains and absent knee-jerks. These facts are very hard to explain, and they support in a measure Raymond, Jendrassik, and Fränkel's view that the incoördination of tabes is due to psychical disturbances. Raymond argues that if co-ordination is a cerebral function, then the incoördination of tabes is a cerebral disturbance. Reference to Fig. 1 shows that in tabes the direct sensory path to the opposite cortex is interrupted by the degeneration of Goll's column. This, however, is only one cause of incoördination. It would certainly lead to defective consciousness of the changes occurring in the tension of tendons and muscles and skin, and in the pressure on joints during movement of the limbs, and volitional impulses would be impaired by this defect of consciousness; but this cannot be the whole explanation, for in general paralysis we have a progressive destruction of the cerebral cortex: yet unless the posterior columns of the spinal cord are affected there is not coarse ataxic incoördination. Pierret has also recorded two cases of sclerosis of Goll's column without ataxy. For the maintenance of bodily equilibrium in the erect posture, while standing or during the successive changes that occur in the trunk and limbs in progression, a proper adjustment in the contraction of correlative antagonistic muscles is necessary. In standing erect, the joints are fixed by the tonic contraction of the antagonistic muscles of the lower limbs. The motor neurons of the anterior horn which preside over the muscles are excited by impulses from the periphery. Unequal and imperfect transmission of sensory impulses will lead to unequal and imperfect excitation of these motor neurons, and to a corresponding unequal and imperfect innervation of the muscles whereby their normal equable tonic contraction is disturbed and lowered. Seeing that in tabes there is a progressive degeneration of the afferent spinal neurons, we can easily understand that there will be a progressively lowered and unequal tonus in the muscles.

Again, the degeneration of the fine plexus around the cells of Clarke's column (*vide* Fig. 7), met with early in the disease, is sufficient to explain the loss of power of balancing the body when the basis of support is narrowed by placing the feet together or standing on one foot, even though there be no paræsthesia of the soles of the feet. Reference to Fig. 1 shows that atrophy of this fine plexus leads to interruption of the sensory afferent impulses to the cerebellum, and puts this organ, which is concerned with unconscious co-ordination and tonic contraction of the muscles engaged in maintaining bodily equilibrium in the erect posture, at a great disadvantage. On closing the eyes another guiding sensation is removed and the instability is increased, there remaining only the sensations coming from the semicircular canals. Some experiments recently performed by Bickel upon dogs show that this animal after section of the posterior roots exhibits ataxy for a short period of time after being placed in a dark room; but compensation soon occurs. Later the semicircular canals were destroyed, and then the animal when placed in a dark room exhibited ataxic gait, which was permanent. The power of compensation in man is necessarily imperfect, because of his erect posture and of the readiness, therefore, of the line of the centre of gravity to fall outside the base.

In walking, the co-ordination of the correlative tonic contractions of the antagonistic muscles in the resting leg is imperfect for the same reasons as above mentioned for standing; but the disturbance of equilibrium is greater, because the weight of the body is resting on a narrower base. The advancing leg is raised from the ground and moved forward by volitional impulses from the cortex cerebri—contraction of certain groups of muscles with stretching of the antagonists takes place. If carried out perfectly, this necessitates the association of consciousness and volition; but consciousness of the sense of position of the limbs in the successive changes that occur during the movements depends upon the continuous and uninterrupted passage to the cortex of impulses from the skin, joints, tendons and muscles of the parts engaged, impressions which, taken together, make up the kinæsthetic sense. A break in this path is, however, only one factor in the production of incoördination; for if there were not the loss of spinal reflex tonus, and interruption to the cerebellar path, there would not be marked incoördination. Volitional impulses produce an increased contraction of one group of muscles acting upon a joint, and stretching of the correlative antagonists; but if there is diminution and inequality of reflex spinal tonus in the muscles there must necessarily be unequal and imperfect action of the antagonistic muscles. This will of itself cause irregular, jerky, and incoördinate movements; but besides this, the muscles being in a state of unequal tension, false impressions will travel up to the cortex with each successive change of position. The ataxic tries to compensate this by vision.

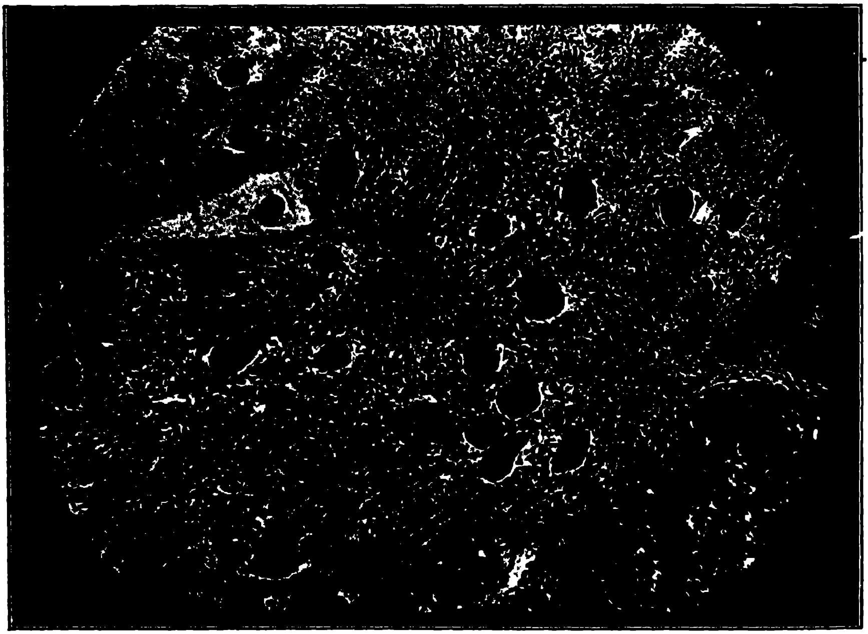

FIG. 7.—Photomicrograph of Clarke's column at the level of the 1st lumbar segment from a case of tabes. There is complete atrophy of the fibres of the postero-external column, which break up into a brush work around the vesicular cells. The cells themselves are unchanged, only the felt work of nerve fibrils which surrounds them is atrophied. Impulses from the periphery are thus cut off from the cerebellum. The axis-cylinder processes of these cells form the cerebellar tract. Magnification, 200 diameters.

The lightning pains may be explained by the degeneration of the root fibres; but their paroxysmal character is difficult to understand. The partial anæsthesia may be accounted for by the partial degeneration of the roots. Professor Sherrington has shown that at least three roots overlap one another in a skin area, and that in order to produce complete loss of sensation in a part all three roots must be divided. If there be pronounced analgesia or anæsthesia the peripheral nerves are probably affected. With regard to the visceral crises nothing is known. It was suggested by Ross that these phenomena might be associated with the degeneration of the nerve network around the cells of Clarke's column. No doubt in the dorsal region sensory fibres come from the viscera by the posterior roots. Dehler's observation already referred to suggests the possibility that in tabes sensory fibres passing from the sympathetic ganglia to the posterior spinal ganglia may be found degenerated; certainly the matter is worthy of investigation.

The Argyll-Robertson pupil is the most frequent objective symptom of tabes, as it is of general paralysis. In the former disease the reflex rigidity of the pupil to light is generally associated with contraction—in the latter with dilatation. No very definite anatomical facts have been brought forward to explain this remarkable phenomenon; it is asserted that it is due to a break in the synapse between the terminal arborisation of optic nerve fibres in the corpora quadrigemina and the dendrites of the sphincter iridis nucleus. The pathology of tabes has been more clearly intelligible with the modern conception of the neuron as an independent complex cell unit; and although there are a great many problems yet to solve before we can satisfactorily explain many of the clinical phenomena, yet the fundamental principle of the neuron has revolutionised our ideas of the pathology of this disease. Sclerosis due to overgrowth of the glia tissue is secondary and proportional to decay and atrophy of the neurons.

F. W. Mott.

Symptoms.—An ordinary case of tabes falls naturally into three stages, according to the degree of intensity in the principal symptom—incoördination. In the first, sometimes called the pre-ataxic stage, there is no incoördination; the patient can stand and walk normally. In the second stage standing and walking are rendered more or less difficult. In the third the patient cannot stand or walk alone.

Probably the earliest symptom to attract the patient's attention will be pain. Of this there may be various kinds, but the most characteristic are the so-called lightning pains. These are sharp and shooting in character, sudden in onset, momentary in duration; they occur in bouts which last a variable time, which are paroxysmal (leaving intervals, it may be long intervals, of freedom), or which may be periodic. The general character, then, of the pains is neuralgic; but they are not, like neuralgia, necessarily limited to special nerve districts; they may haunt one spot, may be widely distributed, or may shift their seat rapidly. Usually the earliest pains are in the lower limbs; but this is not invariable, they may begin in the face or head. They are generally aggravated by damp or by cold winds, so that the patient calls them "rheumatics." They may leave a transient cutaneous hyperæsthesia. Sometimes in connection with lightning pains, and sometimes without them, a sudden giving way of the legs has been noticed. There may be pain, too, that is not lancinating, but has a dull aching or boring character, and is situated in the back, trunk, head, or limbs. Many are the comparisons made by the unfortunate patients in whom the pains are severe: "like knives, needles, electricity"; "like gimlets, corkscrews, or hot irons thrust into the flesh"; "like wild beasts gnawing," and so on.

A history of causeless paroxysmal shooting pains should of itself direct our thoughts towards tabes; but other complaints may be made. These, as we shall see, may be very variable, but some are particularly common: for instance, diplopia, squint, or ptosis, depending on some transient oculo-motor palsy; or difficulties of micturition, so that the patient either cannot pass water readily, or (which is perhaps more frequent) cannot retain it properly; the urine escapes from him involuntarily, or he has to hurry when once he has felt the desire to micturate. There may also be troubles in the genital sphere; or in walking the patient may experience a sense of fatigue quite disproportionate to the exercise.

Luckily the diagnostic importance of such complaints can be tested by physical examination, and for this we turn to the state of the knee-jerks and the state of the pupils.

I need not now dwell on the method of investigating the *knee-jerk* [*vide* art. "Tendon Reflexes," vol. vi.] and of the general inferences to be drawn from it. Suffice it to say that in tabes, even at an early stage, the knee-jerk is commonly absent, while the power, nutrition, and electrical reactions of the extensor muscles of the knee remain normal. This disappearance of the knee-jerk in tabes has been called Westphal's sign. The date of its disappearance we cannot usually fix; in some few cases the knee-jerk may disappear under observation, becoming feeble, unequal on the two sides, and finally absent; in some few it may remain present for years; in some an early stage of exaggeration has been reported; but as a rule, when the patient first presents himself it is gone.

The symptom derivable from the *pupil* consists essentially in this—that the pupil does not contract (as normally it should do) when light is suddenly thrown upon the eye; but the normal contraction still takes place when the patient fixes a near object (that is, accommodates or converges). This is known as reflex iridoplegia, or as Argyll-Robertson's sign. Commonly the pupils are small—sometimes so small as to deserve the name of "pin-point pupils," and symmetrical with each other, both in size and in mode of action. Inequality in size, however, is not very uncommon, though uniocular reflex iridoplegia is decidedly rare. It is said, too, that such pupils do not dilate under the stimulus of pain. Varieties of action occur; thus a pupil may contract to light, and then re-expand though the illumination be still maintained; probably this marks an early stage of reflex iridoplegia. Not infrequently, also, the pupils react neither to light nor to accommodation. The date of the appearance of the pupil sign is as difficult to fix as that of Westphal's sign. In some cases it may not appear at all, the pupillary reactions remaining normal; but on the whole it is a very uniform, trustworthy and early symptom.

The combination of these three symptoms—lightning pains, loss of knee-jerk and reflex iridoplegia—warrants a diagnosis of early tabes; and this is all the more certain if there be added the history or presence of ocular paralysis, difficulties of micturition, or some of the anomalous symptoms which I shall presently describe.

The *second stage* of tabes is characterised by the appearance and the progressive increase of ataxy, generally also by defects of sensation. By ataxy or incoördination we understand a want of harmony or co-operation in muscular contractions (as distinct from loss of power to contract), whether they be employed to produce movement (motor ataxia) or to maintain a fixed position (static ataxia); so that from the ill-combined muscular contractions actions result which are disorderly and inappropriate to their purpose. Incoördination, as distinguished from paralysis, has been recognised ever since Todd's and Duchenne's writings as a special mark of tabes; still, we must remember that a certain amount of loss of power often coexists with it. Incoördination first reveals itself in walking and standing, actions which require the co-operation of many muscles both of the trunk and lower limbs. It begins as a mere unsteadiness in walking, which is most noticeable when the patient rises to walk, or when he turns; or it may be brought out by making him march, halt, or turn to command, walk along a given line, walk backwards, walk up and down stairs, and so forth. Above all, it is worse when he closes his eyes; and often he will volunteer the statement that he cannot walk in the dark. At first the gait suggests a mere loss of equilibrium, such as might be due to an affection of the trunk muscles; but in time the movements of the legs themselves become peculiar, the patient gets the feet crossed as he turns, and he plants them widely apart and uncertainly, looking to see where they are going. Finally, ho manifests the typical "ataxic gait"; he rises from his seat and starts carefully, probably invoking the support of sticks or of a friend; he bends forward at the hips, looking at his feet; he lifts the advancing foot too high, throws it outward with a jerky movement, and brings it down again with a stamp. The action of the lower limbs has been compared to the walk of a cock, or the movement of the legs of a marionette. Even when sitting he is unable well to direct the movements of his legs if he be told to describe a circle with his toe, touch one knee with the opposite heel, and so on. In standing, as in walking, he needs a wide basis for support, hence he is unsteady when his feet are put close together; or even supposing he can stand so, yet on closing his eyes he begins to sway and stagger. This staggering when the eyes are shut is known as Romberg's symptom, and is usually considered an early indication of ataxy. Later the patient finds difficulty in standing at all without support. The incoördination spreads in time to the upper limbs. Here it causes an awkwardness and want of precision in the movements of the hands, so that there is difficulty in writing, in buttoning the clothes, in playing a musical instrument, in carrying out the finer manipulations of trade, and so on. If the patient be told to pick up a pin from the table, the hand wavers, and hovers over it before arriving at it, and the fingers fumble before grasping it. In the upper, as in the lower limbs, the maldirection of movement is much increased when the aid of sight is denied. Thus a favourite test is to tell the patient to touch the tip of his nose quickly with one fore-finger, first with the eyes open (which he will do unless the ataxy is pronounced), next with the eyes closed (when the finger will probably go wrong).

Defects of sensation are usually found by the time that incoördination is well established. The commonest and most obvious of these begin in the lower limbs. The patient has sensations of tingling, pins and needles, numbness and deadness, and when walking may experience a peculiar feeling as if he were treading on cotton-wool or on a thick carpet. In the trunk, too, the well-known "girdle pain" may appear—that is, the sensation of a cord tied tightly round the body. Examination may then show anæsthesia of various degrees, of various distribution, and affecting variously the several forms of sensation (touch, pain, temperature, etc.). To these points we shall recur presently, but it may be stated here that analgesia (readily tested by the prick of a pin) and delay in the transmission of such sensation of pain is particularly common. Sometimes the sense of posture is lost, so that the patient cannot tell how his legs are placed without looking at them, nor imitate the position of one limb with the other when his eyes are closed. Sometimes he is unable to discriminate the difference of weights placed in his hands.

The general tendency of the ataxy is to get slowly worse, and to spread upwards to the hands and arms. The same may, perhaps, be said of the more definite forms of sensory defect. The lightning pains in some cases disappear as the disease advances; the pupil sign and the absence of knee-jerk remain, as a rule, unaltered.

The *third stage* is but an aggravation of the second, in which the increase and spread of the ataxy has rendered the patient quite helpless. But there may, indeed, be a genuine paraplegia, and there is likely to be more definite paralysis of the bladder than before, and sexual impotency, if this have not existed from an earlier stage. Confined, as he is now, to bed or to a chair, emaciated and feeble, the patient's life is obnoxious to peril from bedsores, cystitis, or intercurrent disease.

Having now described what is usually considered the standard form of tabes, I will next consider in more detail the motor, sensory, and reflex abnormalities which occur in this disease; and particularly those affections of the special senses, of the viscera, and of the joints, which often form such striking and dominant features of it as to throw the classical locomotor ataxia quite into the shade.

Motor affections.—I have already alluded to two motor affections other than ataxia: first, a disproportionate fatigue after exercise, which is an early symptom in some cases, and which, according to Pitres, may occur in paroxysms just like the pains; secondly, a true muscular weakness of the legs, appearing alongside of the incoördination. Such weakness is in most cases a subsidiary affection, requiring examination to detect it. For when paralysis and incoördination of the limbs are at all proportionate to each other the case will probably be ranked as ataxic paraplegia rather than tabes, though the line between the two diseases may be difficult to draw. But it is more important to notice the many forms of genuine motor paralysis which may present themselves; and these, from their very variety, it is difficult to classify in an exact and useful manner. First, let me say that some of them may occur early in the disease, are limited in their area, and transient in nature. Such is usually the character of the very common ocular paralyses; thus, in quite the early stages of tabes one third nerve or a branch of it, one sixth, or one superior oblique, may be paralysed partially or totally, and may recover again in a few months, weeks, or even days. But even these ocular palsies may be bilateral; thus double ptosis has been frequently noted, and paralysis of both sixths sometimes; nor do they invariably recover. Less commonly the early paralysis takes the form of an hemiplegia or a paraplegia, so that a patient may suddenly lose power in both legs, or in the limbs of one side, and recover; but the recovery is followed by the symptoms of tabes. Such early transient palsies are alleged by some authors to be genuinely syphilitic, and, indeed, gummata and arterial disease have sometimes been found after death; still it must be remembered that similar palsies occur in the early stages of disseminated sclerosis, a disease with which syphilis has nothing to do. While speaking of hemiplegia we may note two other facts: first, that tabes may be heralded or accompanied by sundry other cerebral phenomena, such as fits, either epileptic or epileptiform, or attacks of vertigo or coma, recalling the much more common occurrences of this kind in the allied disease general paralysis of the insane; secondly, that, apart from the early and recoverable hemiplegia, hemiplegia of a more permanent character, and depending probably on ordinary vascular lesions, may occur in any stage of tabes. But to recur to palsies limited to the distribution of nerve-trunks. These may occur, though less commonly, in other districts than the ocular; particularly in that of the peroneal nerve, or in that of the ulnar, radial, or spinal accessory. These nerve-palsies may be either single or bilateral. Bilateral laryngeal paralysis I shall discuss later. Paralysis in the district of the hypoglossal is marked by atrophy of one-half of the tongue; the affected lateral half of this organ is flabby, furrowed longitudinally, and wasted; when protruded the tip points to the paralysed side, and the paralysed muscle cannot be felt to contract; nevertheless, the patient finds singularly little inconvenience either in speaking or swallowing. It would seem that such lingual hemiatrophy is due to disease sometimes of the nerve, sometimes of the nucleus. The mention of nuclear disease brings us to yet another class of palsies of a more generalised and progressive nature than those we have been considering, clinically resembling progressive muscular atrophy, and like it caused either by multiple neuritis or by disease of spinal and bulbar nuclei. Such progressive disease may begin with wasting of the intrinsic muscles of the hands, and spread thence to the shoulder and other muscles; or in the lower limbs; or it may begin in the tongue and lips taking the form of a progressive bulbar paralysis; or in the muscles of the eyeball as a progressive ophthalmoplegia. This combination of tabes with progressive muscular atrophy is not, however, common [*vide* "Bulbar Palsies," p. 219].

Sensory affections.—Only a few authors have made systematic and exact observations upon the sensory defects of tabes. It has been recently stated that a diminution of sensibility in the trunk is a common and early symptom, so that the patient, though not really anæsthetic, cannot feel light touches here. This dulling of sensation, "hypæsthesia" as it has been called, affects (it is said) a zone corresponding to the middle and lower dorsal nerves; but it may spread thence and reach the limbs. At the edges of the zone there may be hyperalgesia, especially when the stimulus of cold is applied. In the limbs, on the other hand, particularly the lower limbs, there may be more definite anæsthesia. This too may sometimes be found in the face and head, even where there is none in the limbs. All modes of sensibility—tactile, painful, thermal, etc.—may be abolished together, or there may be "dissociation" as it is called, that is to say, the several modes may be very unequally affected. Where there is such dissociation sensibility to pain is particularly apt to be lost; but a special thermal anæsthesia is far less common in tabes than in syringomyelia. The analgesia may affect not only the skin, but also deeper parts, such as the muscles and bones; analgesia upon pressure of the testicles has been specially noticed. According to Oulmont, tracts of cutaneous anæsthesia are symmetrical when they affect the limbs, but may be unilateral when they affect the face and head; and they have certain favourite sites, such as the toes, plantar surface of feet, ankles, knees, certain points about the trunk or fingers; later authors, however, doubt this latter statement, and probably we cannot yet speak very definitely about their distribution. In the upper limbs anæsthesia is said to begin in the ulnar fingers and ulnar side of the hands and arms. Analgesia of the ulnar nerve has been particularly noticed. A healthy man, when his ulnar nerve is forcibly compressed against the bone just above the inner condyle of the humerus, feels numbness and tingling in the little finger—the ordinary "funny-bone" sensation—and pain at the point of compression; but a patient with tabes (it is said) feels the peripheral tingling, but not the local pain. Lastly, short of downright anæsthesia, and sometimes as the precursor of it, perversions and impairments of sensibility, of very various kinds, often occur. Thus sensory impressions (and particularly, as I have said, painful impressions) may be delayed in transmission, so that the patient does not cry out till some seconds after he is pricked or pinched; or he may feel the contact of the pin-point at once, but the pain of the prick only after a distinct interval. Or a touch may be felt, but wrongly localised. Or he may feel that he is touched, but be unable to say with what sort of thing he is touched. Or he may be insensitive to slight stimuli, but unduly sensitive to moderate stimuli of the same nature; and so forth.

Affections of reflex actions.—The cutaneous reflexes are comparatively unimportant in tabes; it would appear that they correspond generally with the tactile sensibility of the skin, being absent when the skin area from which they are produced is anæsthetic, and present when it is not. It is said that the cremasteric reflex is absent when the patient is impotent. Of the organic reflexes, difficulties of micturition, such as slowness and straining, or imperfect control over the bladder, are (as I have said) frequent in the early stages; paralysis of the bladder may occur later, but is not perhaps so frequent as might have been anticipated. The function of defæcation is less frequently affected. The sexual function in the man is prone at first to abnormal irritation; Trousseau remarks that a patient with early tabes may be capable of repeating the sexual act several times in a single night. Impotence with absence of desire supervenes sooner or later. Women who are the subject of tabes have been known to bear children; but it is certainly more common to find a history of sterility, whether this be due to the tabes or to the conditions which lead up to it.

The very great importance of the tendon reflexes in diagnosis I have already pointed out. The early disappearance of the knee-jerk is due to the fact that the spinal degeneration, attacking the fibres of the posterior roots soon after their entry into the cord, usually begins in the lower dorsal and lumbar regions, and thus cuts the reflex arcs which pass through this part. In those rare cases where the degeneration begins higher up in the cord (cervical tabes) the knee-jerk may be retained. The disappearance of it is therefore a sign of local disease; namely, of a lesion in the postero-external column of the dorsi-lumbar region. If this part be completely destroyed the knee-jerk will be completely absent, whatever the other conditions may be; but, if the destruction be incomplete, it is possible that the knee-jerk may be maintained, or even exaggerated if there be simultaneous degeneration of the lateral columns. This may be seen in cases of combined postero-lateral sclerosis; and similarly, in some cases where hemiplegia has occurred in the course of tabes, the knee-jerk of the paralysed side has been temporarily restored by the descending lateral sclerosis which results from the cerebral lesion. On the other hand, when the posterior sclerosis is advanced we may witness in such hemiplegic cases the curious combination of post-hemiplegic rigidity with absence of knee-jerk.

Affections of special senses.—Among affections of the special senses loss of smell or taste and subjective sensations of smell or taste have been described; but they are certainly infrequent. Auditory affections occur oftener—either vertigo or, what is more common, deafness; such deafness is, I believe, generally incomplete and bilateral; and its cause may be difficult to determine. In one well-known case, indeed, there was absolute deafness, and after death atrophy of the auditory nerves was demonstrated, quite analogous to the better known optic atrophy. But it would be a mistake to suppose that deafness is always due to atrophic changes in the nerve. Sometimes, certainly, the cause lies mainly in the middle ear; for inspection shows an opaque hillocky retracted membrane, indicating a fibrosis of the tympanum; in other cases examination with the tuning-fork points either to "central" mischief (which of course may be either in the labyrinth or in the nerve), or to a mixture of central and middle ear mischief. It has been supposed that chronic fibrosis of the middle (and inner?) ear in tabes depends on some perverted trophic influence in connection with the fifth nerve; but obviously a certain allowance must be made for mere coincidence, since chronic middle ear catarrh is in itself a common affection.

Affections of the eye are far more important, and foremost among these stands atrophy of the optic nerve. The frequency of this has been estimated at 15 per cent or thereabouts. It generally begins at an early stage of the tabes, preceding the ataxy, nay, sometimes preceding the pains, so that such cases may come first under the care of the ophthalmic surgeon. The patient notices a failure of vision, first in one eye and, after a variable interval, in the other; and this as a rule progresses to complete or nearly complete blindness in both eyes. Commonly, as his visual acuity diminishes, the patient becomes colour-blind, and his fields of vision become contracted, either concentrically or irregularly. Sometimes large sectors of the field disappear, and this may even produce hemianopia. Central scotoma appears to be very rare. The defects in the two eyes, though they may differ as to their stage of degeneration, have a general symmetry both in their distribution and their progress. Visible changes in the disc begin to appear by the time that loss of vision is complained of, and sometimes even in the eye which is not known to be failing. The changes mainly consist in pallor, gray discoloration, opacity, and undue sharpness of outline. As the vascularity of the disc diminishes, as its medullated nerve-fibres atrophy, and as its interstitial tissue increases in amount, so it loses its rosy translucent aspect, and becomes pale and opaque, taking on usually a dead gray colour, or sometimes a yellowish tint, or even a chalky white. The physiological cup may appear deep, with undue exposure of the lamina cribrosa, or it may be filled up, so that the surface of the disc looks flattened. The edge of the disc is sharp and clear-cut all round, so that it contrasts with the surrounding fundus. The retinal vessels may remain of normal size. Upon the great prognostic importance of optic atrophy, involving as it does the prospect of utter blindness, it is unnecessary to insist. The gloomy outlook is somewhat relieved by a belief, which is commonly entertained, that when tabes begins with optic atrophy the ataxic symptoms are likely to be less marked and less progressive than usual. Neither must we forget its diagnostic importance as an early sign of tabes; for primary progressive optic atrophy is comparatively common in this disease and rare as an isolated occurrence; therefore, when a patient consults us for this alone, a careful search must be made for other symptoms.

The principal abnormalities of the pupil and the very common palsies of the external ocular muscles have already been mentioned. Less common and more serious is a progressive ophthalmoplegia externa. One after another the ocular movements become limited and abolished, till both eyeballs are reduced to permanent immobility. This condition usually comes on early in the disease, and does not pass off. It is due to disease of the oculo-motor nuclei, and may be associated with muscular atrophy in the limbs. While such ophthalmoplegia is progressing, the movements of the eyeballs may be jerky and irregular; but a well-marked nystagmus, such as is so common in disseminated sclerosis, is very rare in tabes.

We now come to the subjects of visceral disturbances and of trophic lesions. Many. of these form extremely striking phenomena, which, although fairly common, have little apparent connection with spinal disease. Hence, when they manifest themselves early in the course of tabes (as they often do), and apparently as isolated facts, their true signification is apt to be overlooked.

Visceral crises.—The so-called visceral "crises" must necessarily arrest attention. These consist in a sudden and violent disturbance of function, for which no sufficient external cause can be found, followed by an equally rapid return to normal, and a recurrence of the attack after a variable interval. This paroxysmal character of the "crisis" suggests an analogy to the bouts of pain, and, indeed, a typical crisis is accompanied with pains. The commonest is the "gastric crisis." In this the pains concentrate themselves upon the epigastrium, nausea sets in, and soon vomiting too of a most severe and intractable character; the patient brings up first his food, then bile-stained mucus or clear fluid. Yet the tongue is clean, and, though the pulse may be frequent, the temperature remains normal. Severe gastric crises may be accompanied by much nervous depression, or even by collapse. After a variable time, usually one or more days, the vomiting ceases as suddenly as it began, leaving the patient well except for exhaustion. But the attack will recur, either at irregular intervals or in some cases with remarkable periodicity. In some attacks there is no pain, only the paroxysmal and causeless vomiting. Usually the gastric crises occur early in the disease, and pass off as it progresses; and later the patient, seeing no connection between them and the paralysis which has since overtaken him, may not speak of them to the physician, or may merely mention that he is subject to "bilious attacks." Conversely, at the time of their occurrence they may form his sole complaint, so that in any case exhibiting causeless and recurrent vomitings the possibility of an early tabes should be remembered.

Analogous to gastric crises, and sometimes associated with them, are the intestinal crises, consisting of paroxysmal attacks of diarrhœa with or without abdominal pains. Sometimes in the rectum the patient has attacks of tenesmus, pains, or other disagreeable sensations. Similar pains and difficulties may arise in the region of the kidney, urethra, and bladder, so as to raise the suspicion of a calculus. In the sexual sphere men may be troubled with causeless erections and emissions; and women with erotic sensations which have been termed clitoridean crises. Anomalies of secretion may accompany, or may constitute these visceral crises. Thus during a gastric crisis the secretion of gastric juice is stated to be increased, and the chlorine of the urine correspondingly diminished; and in the intestinal crisis there is a watery secretion from the bowel. Paroxysmal fluxes of urine, of saliva, of tears, paroxysmal sweatings, and even attacks of glycosuria have been observed.

Affections of the larynx.—Affections of the larynx occur; the commonest of which is a bilateral paralysis, involving the abductor muscles only. The power of dilating the glottis is gradually lost; first of all the cords, which during inspiration should diverge widely, diverge imperfectly or remain immobile during this act, though during phonation they still approximate properly; next they take up permanently a position nearer and nearer to the middle line, till the glottis is reduced to a mere chink. The symptoms of this paralysis are in its earlier stages almost nil; for the voice is unaffected (the tensors and adductors acting normally), and the only difficulty experienced by the patient is in getting breath quickly after prolonged expiratory efforts, as in speaking or singing; but in time there arises a certain stridor during respiration, evident either in the pauses of his conversation, or as a snoring noise when he sleeps; finally, as the glottis becomes extremely narrow, he finds himself liable to attacks of severe dyspnœa on the occasion of any slight laryngeal catarrh, or on extra respiratory exertion, inhalation of cold air, or other source of irritation to the larynx. Probably many of the attacks which are called "laryngeal crises" are really mere exacerbations of a pre-existing abductor palsy; but it would appear that paroxysmal attacks of cough and dyspnœa, analogous to the gastric crises, may occur without other basis than increased reflex excitability or spasm pure and simple. In some instances such laryngeal crisis is accompanied by coma and convulsions, suggesting the possibility of dangerous cerebral complications; this has been called the "laryngeal ictus." A patient with severe abductor palsy is evidently in constant danger, and a tracheotomy, if not performed as a prophylactic, may be required at any moment; yet it is surprising how little inconvenience some patients may experience even when the glottis is considerably narrowed.

Other laryngeal phenomena are unilateral paralysis, affecting movements both of abduction and adduction of one vocal cord (as in ordinary paralysis of the recurrent laryngeal trunk), which, according to Marie, is often associated with paralysis of the soft palate and hemiatrophy of the tongue on the same side; and ataxia of the larynx, an extremely rare condition, in which the cords move, but without proper rhythm or regularity, and there are sudden changes in the pitch of the voice. In some cases difficulties of deglutition occur which may be put down to abnormal innervation of the pharyngeal muscles, and are called "pharyngeal crises."

Circulatory system.—Frequency of the pulse was noticed by the early writers on tabes as a common symptom; less notice has been taken of this lately, possibly because the sympathetic nerve is no longer thought to be concerned in the pathology of the disease. Attacks simulating angina pectoris sometimes occur and have been classed as "cardiac crises." Organic valvular disease, and particularly of the aortic valves, is comparatively common; and elaborate hypotheses have been advanced to account for this; but it is probable that in some cases (looking to the age and sex of the patients) such aortic disease is a pure coincidence, and that, in others the tabes and the cardiac lesion have a common cause, namely, syphilis. Lastly, the symptoms of Graves' disease have sometimes been observed to coexist with those of tabes.

Trophic lesions.—Of trophic lesions the joint disease is the most remarkable. The characters of this were laid down by Charcot, and it has been named Charcot's disease. It occurs most commonly, he maintains, in the earlier stages of tabes, that is, after the onset of the pains and before the onset of the incoördination; apparent exceptions to this statement, such as the occurrence of joint disease in the upper limbs late in the course of tabes, may be explained on the ground that the spinal disease may be of long standing for the lower limbs when it has only just reached the upper ones. Though any joint may suffer, the large are more often attacked than the small, and the knee oftener than any. The affection may begin quite suddenly without any prodroma, or without any exciting cause; but sometimes it may be heralded by lightning pains in the neighbourhood of the joint, or a crepitation on movement may have been noticed; or sometimes there may have been some trivial antecedent injury. The joint becomes suddenly swollen, and on examination presents signs of a large effusion. Frequently this effusion is not limited to the joint itself, but the whole limb in its neighbourhood is firmly œdematous. In spite of its acuteness this process gives rise to no heat, nor tenderness of the parts, nor any pain. In some mild cases the effusion subsides gradually, and leaves little trace behind it; more commonly on its disappearance the joint is found to be completely wrecked. This disorganisation depends mainly upon absorption of the tissues, whether ligaments, cartilages, or bones of the articulation, whereby may be produced either an extreme and abnormal mobility of the joint, so that the limb can be flung about like a flail, or else dislocations of an extraordinary character. But sometimes new bone may be thrown out, to be felt in the form of spicules, bosses, and buttresses, and may produce restrictions of movement. It is rare for the original effusion to suppurate.

The clinical course of this arthritis differs so evidently from that of rheumatoid arthritis, that it seems at first sight surprising that the two affections should have been identified. Yet this identity has been upheld, and particularly by English surgeons. Two facts may be mentioned which, perhaps, have contributed towards this opinion: first, that rheumatoid arthritis, pure and simple, may occur in the course of tabes, as in that of any other chronic disease; secondly, that, judging solely from their anatomical results, it may be difficult in some cases to distinguish Charcot's disease from rheumatoid arthritis. Without entering here into pathological details, it may be broadly stated that extensive and rapid destruction of tissue, particularly of bone, with osteophytic deposits in the capsule and synovial membrane, forming eventually spikes and sheaths of calcareous matter around the joint, mainly occurs in Charcot's disease; while slow thickening, eburnation, and outgrowths of bone, with "lipping" of the cartilages, occur mainly in rheumatoid arthritis.

Two other hypotheses concerning this joint disease have been advocated: first, that it is traumatic in origin, caused either by the ataxic movements of the limb or by the accidents to which an ataxic patient is necessarily liable—this is sufficiently refuted by the fact that it often arises before ataxy has set in; secondly, that it is simply a syphilitic arthritis: for this latter opinion I believe there is little support, neither does it explain the peculiar characters of the joint disease in question. We therefore adhere to Charcot's original view, that the nervous lesion is essentially the cause of the arthropathy, though how this cause acts we do not yet know (vol. vi. p. 557).

Closely allied, no doubt, to the phenomena of the joint disease is that of spontaneous fracture of bone. Such fracture may occur either on very trivial injury or without apparent cause; and, like the joint lesion, is painless. The long bones, particularly those of the lower limbs, are most often broken; but multiple fractures, involving many bones, may occur in the same patient. This last fact of itself suggests some constitutional cause; and it would appear that in the bones generally an absorption of the osseous material has been going on, with dilatation of the Haversian canals, and (in the case of the long bones) of the central cavity of the shaft. The thinning and fragility of the compact substance thus brought about eventually results in fracture. Such fractures may occur in the immediate neighbourhood of diseased joints, indicating, perhaps, that both phenomena may depend on the same cause. So too rare cases of spinal distortion have been described, due partly to fracture of the vertebræ, partly to intervertebral arthropathy.

By a similar combination of bone and joint disease may be produced a peculiar distortion of the foot; first, a firm, painless swelling forms upon the dorsum, subsequently the inner border becomes thickened, the arch is flattened, and the whole foot shortened and deformed. This condition differs from the club-foot described by Joffroy, which is a kind of equinovarus with flexion of the toes, produced (it would appear) partly by a neuritis which has caused muscular palsy and atrophy, partly by pressure of the bed-clothes upon the flaccid and paralysed feet.

Another affection of the lower limbs, possibly trophic, which has been seen in some cases of tabes, is rupture of the tendo Achillis.

Perforating ulcer of the foot is not very uncommon. This begins as a corn, seated commonly, though not invariably, on the ball of the great or little toe; beneath this, or in the centre of it, suppuration takes place, so that an ulcer or sinus is formed, which in bad cases may extend right into the joint beneath. It is common to find some anæsthesia in the neighbourhood of such an ulcer. Gangrene of the toe occasionally supervenes, and either for this cause or on account of the inconvenience entailed by the presence of an intractable sore, amputation may be required. Nevertheless, the less severe forms of perforating ulcer, which are also the commonest, often heal by themselves, or under very simple remedies. [See article on "Perforating Ulcer," etc., vol. vi. p. 572.]

Deformities and loss of the nails, chiefly of the big and little toe, have been seen in tabes, and have been associated with the nervous disease on account of the prevalence of pain or of anæsthesia in the parts concerned. The teeth in some cases have been observed to drop out; and even large pieces of the jaw-bone to come away, painlessly.

As to cutaneous affections, herpes sometimes occurs, preceded by lightning pains in the district of the eruption; sometimes also crops of small subcutaneous ecchymoses, also preceded by pains; sometimes, again, widespread effusions of blood under the skin. Subcutaneous œdema has already been mentioned in connection with the joint disease; it may possibly occur alone.

Mental affections.—In most cases of tabes the mind is not affected. It is true that various psychical troubles, particularly in relation to the early stage of the disease, have been described by Fournier, but these I think are hardly a matter of general experience. Other authors have thought that the contented and hopeful way in which many patients bear their terrible disease is in itself an evidence of some mental alteration. The opposite bias, namely, towards undue despondency and hypochondriasis, though less common is far from unknown. But it is of importance to remember that in the course of tabes symptoms of general paralysis of the insane may supervene; which is hardly surprising when we remember the very close relations between the two diseases. Both occur with greatest frequency in syphilised subjects, and in men rather than women. In general paralysis there may exist, over and above the cerebral lesion, a systematic spinal sclerosis, sometimes predominating in the posterior columns; and in such a case the patient may be ataxic, and present the reflex iridoplegia and the absence of knee-jerk which are characteristic of tabes. So that it has been even asserted that the two diseases are essentially the same, and that general paralysis is a "tabes of the brain." Without subscribing to such a view, and holding rather that in ordinary tabes mental symptoms are somewhat uncommon, we have yet to remember that they do sometimes supervene in the form of exaltation, excitement, and the grand delusions characteristic of general paralysis. With the onset of such a mental state the walking powers may appear to improve. The prognosis is, no doubt, very grave, still the mania does in some instances pass away and leave the patient in his previous state; more commonly, however, he gets rapidly worse both in mind and body, and dies a helpless and emaciated general paralytic.

Course of the disease.—The ordinary course of tabes consists in a slow but steady progress, the powers of locomotion growing worse by degrees, and the incoördination spreading to the upper limbs. The several stages are to be measured by years, or at any rate by many months. Not infrequently, however, a natural arrest appears to take place; for patients may suffer pains for many years without becoming ataxic; and others already ataxic may be seen going about for an indefinite time. Many symptoms, such as the early palsies, the pains, and the visceral crises, may disappear as the disease advances; but thoroughly established ataxia rarely, if ever, disappears, though instances of improvement may be quoted. Neither does the knee-jerk return when once completely abolished, nor the pupil regain its normal contractility. It is, indeed, obvious that when the fibres of the posterior roots and columns are destroyed they are never likely to be replaced; nor is it easy to see how their functions can be undertaken by other parts, and therefore such symptoms as depend on this lesion will persist. Still many symptoms may be caused in other ways, and particularly by peripheral nerve-disease, and then may be far less hopeless. On the whole we may say that, the more typical and steady the march of the disease, the less the chances of improvement in the particular case.

Tabes does not necessarily shorten life, and doubtless many patients outlive their physicians; but danger may be anticipated in particular cases from laryngeal, cardiac, or cerebral complications; or in the later stages from bedsores or bladder troubles, or (which is still more likely) from intercurrent disease.

It is unusual for tabes to run a rapid course; still patients have been known to become ataxic in a few weeks or months, or even, it has been said, in a single night.

There are cases in which the pains, anæsthesia, and ataxia affect the upper limbs first; or in which symptoms first appear in the districts of the cranial nerves. In these (which have been called "cervical" or "cerebral" tabes respectively) the sclerosis presumably begins in the upper parts of the cerebro-spinal axis, not as usual in the lower. The diagnosis then may be far from easy, especially in view of the fact that the knee-jerk is likely to remain unaffected longer.

It is important to remember that many of the anomalous symptoms which I have described may occur quite early in the disease; this applies particularly to the optic atrophy, the visceral crises, the joint disease, perforating ulcers of the feet, and abductor paralysis in the larynx.

General considerations.—As we have now described the course, symptoms, and pathology of tabes, I propose, before turning to diagnosis and treatment, to make some general remarks on the disease. The study of tabes has been facilitated by the fact, that both during life and after death we can generally arrive at a tolerably definite diagnosis. For though its manifestations may be very varied, yet there is nearly always a substratum of certain cardinal symptoms; and anatomically it presents a very definite lesion, namely, symmetrical degeneration of certain portions of the posterior columns, and of the posterior nerve-roots. This degeneration is in a double sense progressive: first, because it follows from the laws of secondary degeneration that when fibres have been destroyed by disease at any one level, their prolongations into the upper levels of the cord must perish also; secondly, and chiefly, because the primary process is itself progressive, and so far from limiting itself to one level tends to attack, in upward series, the roots or root-zones of many levels. But although the characteristic lesion, without which we could hardly reckon a given case as tabes, be degeneration of the posterior columns, it is likely that the morbid process has a wider incidence. This is rendered probable by the variety and extent of the symptoms, and is corroborated by the facts of optic atrophy and of peripheral nerve degeneration in tabes. Tabes from this point of view has been called a "great sensory neurosis"; that is, a disease which selects for attack the afferent nervous apparatus, whether central or peripheral. As such it may be contrasted with amyotrophic lateral sclerosis, a disease which falls specially on the efferent part of the nervous system. This view is presented with still more definiteness by Marie, who holds that the afferent fibres degenerate in consequence of disease (not yet demonstrated microscopically) in the cells which form their centres of nutrition; namely, (i.) the cells in the ganglia of the posterior roots, with which the fibres of the posterior roots and posterior columns are connected; and (ii.) the cells of the peripheral sense organs with which the fibres of afferent nerves are connected. Whatever be the precise point in which the degeneration originates, it certainly affects afferent structures mainly and most frequently. Still, it is not entirely confined to them, as is shown by the frequent occurrence of paralysis, and by the facts that degeneration has been demonstrated in motor nuclei and motor nerves also. Such a widely-spread process suggests a constitutional cause, and this consideration leads us to doubt the inferences which attribute the origin of tabes to purely local lesions, such as syphilitic meningitis, syphilitic arteritis, traumatisms, and the like. The view which refers the degeneration to some chronic blood-poisoning, which has originated in most cases from bygone syphilis, in some cases from antecedents as yet unknown, covers the facts better, and can be supported, as we have already said, by the analogy of post-febrile and toxic palsies; but it is as yet without direct proof.

The symptoms cannot all be satisfactorily traced to their anatomical origin. Todd, who first described the symptom of incoördination, connected it with disease of the posterior columns; and in tabes we think there can be little doubt that this connection holds. Since the posterior columns are mainly composed of fibres from the posterior roots, and therefore mainly of afferent fibres, it is rational to conclude that ataxia in general depends on interference with afferent nerve impulses. This view has been energetically contested; yet it receives considerable support from the fact that ataxia without paralysis is sometimes seen in peripheral neuritis and in disease of the posterior nerve-roots external to the cord itself. The lightning pains are commonly attributed to irritation of centripetal nerve-fibres within the cords (namely, the "bandelettes externes," which Pierret has shown to suffer early in the disease); but it is possible that in some cases peripheral neuritis may also contribute to the production of pains.

The loss of knee-jerk Westphal refers to degeneration of the posterior root-fibres in the dorso-lumbar region, just after their entry into the cord. Ross considered that the reflex iridoplegia is due to disease of fibres connecting the anterior corpora quadrigemina with the nuclei of the third nerves; but this view has not been universally accepted.

The explanation of many of the accessory symptoms (visceral, trophic, etc.) is still uncertain. Dr. Buzzard, observing that gastric crises and joint disease frequently occur in the same patient, suggests that both may be due to some lesion in the neighbourhood of the vagal nucleus, where may be placed centres which control the functions of the stomach and preside over the nutrition of the bones and joints. Another explanation of symptoms of this class is, that they depend on peripheral neuritis, and that the occurrence of several such symptoms in the same patient may indicate a variety of the disease in which the peripheral nerves are specially prone to suffer.

The diagnosis of tabes may often be made before ataxy sets in; and a well-established case of ordinary tabes is certainly not easily mistaken, except it be for certain cases of peripheral neuritis. Most cases of *peripheral neuritis* exhibit, it is true, paralysis rather than ataxy, accompanied usually with some muscular wasting and changes in electrical reaction; and such features form a sufficient contrast to ordinary tabes. But sometimes peripheral neuritis produces ataxy without distinct paralysis, and since there may be pains and sensory defects, and since the knee-jerks may disappear, much difficulty of diagnosis may arise. Yet a diagnosis must be made if possible, for the chances of recovery are far greater in neuritis than in tabes. The points most useful for this purpose are as follows:—For peripheral neuritis it is usual to find some immediate cause; either some toxic agent such as alcohol, lead, or arsenic, some antecedent fever, particularly diphtheria, or some constitutional affection like diabetes; for tabes this is not the rule. Peripheral neuritis increases with comparative rapidity, so that the ataxic stage is reached probably in a few weeks or months, and the affection may spread quickly to the arms; in tabes there is a long preliminary stage, and the upper limbs may escape long after the walking powers have been impaired. Accessory symptoms, such as visceral and trophic disturbances, are far more common in tabes. Lastly, the state of the pupil is of extreme importance. Where there is distinct and definite iridoplegia we may generally diagnose tabes; but when the pupil reacts normally, or is fixed, reacting neither to light nor accommodation, the diagnosis in default of other evidence must remain doubtful.

The *diagnosis from general paralysis* is equally important, for here the prognosis (at least *quoad vitam*) is worse than in tabes. Between the general paralysis that begins with characteristic mental symptoms and ordinary tabes there can be little risk of confusion. But there may be a spinal type of general paralysis, in which the patient exhibits ataxia, loss of knee-jerk, reflex iridoplegia, and perhaps other symptoms which we have described as belonging to tabes. We must then look carefully for signs of mental degeneration, such as early loss of memory, irritability, hebetude, and business incapacity (which I believe to be exceptional in pure tabes), and particularly for the incipient affection of speech, and tremulousness of tongue, lips, or hands, which characterise general paralysis. In general paralysis the progress of the disease is generally more rapid than in tabes, just as we expect its conclusion to be. Absence of lightning pains, from which most patients with tabes suffer at some time, should also suggest the possibility of general paralysis.

The name of "*ataxic paraplegia*" has been given to cases in which the lateral as well as the posterior columns are degenerated, and indicates the leading clinical fact, namely, that here paralysis of the legs, usually of a spastic type, is associated with ataxy. Commonly the knee-jerks are exaggerated, since the posterior root-zones of the lumbar region are not completely sclerosed. But sometimes the posterior sclerosis predominates over the lateral, and the clinical picture may then approximate closely to that of tabes, and indeed the distinction becomes a matter chiefly of pathological interest (*vide* p. 145).

Ataxia from *cerebellar disease* is characterised by a staggering, tipsy gait, often with a tendency to fall to one particular side; jactitation of the feet, like that seen in advanced tabes, has been described, but certainly is not the rule. The knee-jerks, too, are occasionally absent in cerebellar disease. Here the resemblance to tabes ends, for there is neither reflex iridoplegia, nor lightning pains, nor other sensory symptoms. The same may generally be said of Friedreich's form of "hereditary ataxia"; moreover, the age at onset, the family history, the affection of speech, and sundry other data will aid us here.

An ataxic gait is sometimes seen in disseminate sclerosis; but the knee-jerks are usually exaggerated, the pupils act normally, and nystagmus, so rare in tabes, is common; while lightning pains, common in tabes, are rare here.

In *syringomyelia* the lower limbs may be ataxic, but are more commonly paraplegic; the disease usually manifests itself first in the upper limbs and upper parts of the body; sensation is affected in a special manner, the temperature sense being first abolished; muscular atrophy, spontaneous whitlows and necroses of the phalanges, and lateral curvature of the spine are common, while in tabes they certainly are not.

Hysteria does not counterfeit tabes; for where the knee-jerk is absent, and the pupil does not react to light, hysteria may be so far excluded; and, on the other hand, where both these signs are absent, tabes cannot be certainly diagnosed.

Treatment.—Concerning treatment we are not in a position to make very definite and satisfactory statements. There are difficulties, obvious enough, in the way of estimating results; for the progress of the disease is slow, nay, sometimes it seems to undergo natural arrest, so that long observation is necessary before we can assert that a remedy has done permanent good. On the other hand, even as the disease advances, some of the most striking symptoms, such as lightning pains, gastric crises, and the like, may spontaneously disappear, whatever the treatment may have been. But in spite of such sources of fallacy we are bound to consider how suffering may best be mitigated, and arrest of the disease procured.

For the pains I consider antipyrin, and drugs of this class, to be fairly trustworthy remedies. Ten grains of antipyrin, or an equivalent of antifebrin, repeated hourly for three or four doses, will often cut short a bout of lightning pains. Or, if the pains be less paroxysmal, the drug may be given regularly three or four times daily. Salicylate of soda, colchicum with alkalies and with iodide of potassium, and aluminium chloride are also drugs that may be used in cases where the pains are a prominent feature. When the pains are very severe and intractable, subcutaneous injections of morphia must be given, but the great danger of creating a morphia habit must be borne in mind. Gastric crises and other paroxysmal visceral disturbances may sometimes be controlled by antipyrin and its congeners. For the urinary derangements ergot has been particularly recommended by Charcot. Looking upon the disease as a progressive degeneration of the nerve-centres, we shall advise the patient to aim at some quiet, wholesome mode of life free from anxieties and from the drive of excessive business; and to abstain carefully from excess in alcohol, smoking, and sexual indulgence, if indeed he be still sexually capable. Remembering, too, that the pains are intensified by cold and damp, we shall advise him to live in some dry and healthy situation, sheltered from east winds, and to winter abroad if he can afford to do so. The general nutrition must receive careful attention—iron, cod-liver oil, and other tonics being used as required, and a wholesome, nutritious diet insisted upon; for patients with tabes are generally pale and thin, sometimes actually wasted. Prolonged confinement to bed is not advisable, except under special circumstances; it is better that the patient should continue to practise the use of his limbs.

Of hydropathic treatment some authorities speak highly, others with reserve; and it would seem that no vigorous or extreme measures should be attempted in this direction, nor too much expected in the way of relief.

Electricity has been applied in two ways: first, faradism, applied to the skin with the wire brush, chiefly with the view of relieving pain, of restoring sensation to anæsthetic parts, and, possibly, of acting upon the spinal cord in a reflex way; doubtless static electricity might be used for the same ends; secondly, galvanism applied to the spine so as to reach the cord, if possible, and to modify its nutrition; a current being used of as many milliampères as can be borne. Two very large electrodes should be used, and placed the one over the sacrum, the other higher up. The current is gradually raised to the maximum, and the upper electrode moved slowly up and down the spine so as to vary the density without rapid makes or breaks. Lastly, electrical treatment may be very properly employed for the various paralyses which occur in the course of tabes, being applied to the affected nerves and muscles in the ordinary way.

As to drugs, considering the prominence assigned to syphilis as a cause, it might be thought that mercury would take the first place. Yet there is little conclusive evidence in its favour, and some authors think it positively harmful. Iodide of potassium, on the other hand, certainly seems to be of benefit in some cases, probably not so much as an antisyphilitic as by some more general action. Sir R. Gowers, who thinks that much may be done by the persevering use of appropriate drugs, speaks highly of arsenic; and, where there is much pain, of aluminium chloride. The old-fashioned treatment by nitrate of silver, administered in small doses for a long time, is capable, I believe, of doing some real and permanent good.

Various mechanical and surgical methods of treatment have been tried. The mildest of them is the gymnastic method, employed to relieve the ataxy. The patient is made, under medical supervision, to practise systematic movements, beginning with the simplest, such as deliberate flexion or extension of a joint, and proceeding to the more complex, such as touching given points with the foot, describing figures, and, finally, balancing and walking. The upper limbs, if ataxic, are to receive a similar education. This plan, which is capable of considerable elaboration, is said by continental physicians very greatly to relieve the difficulties of co-ordination. Other modes of treatment consist in the application of counter-irritants—setons, blisters, and particularly the actual cautery—to the spinal column. Stretching of one or both sciatic nerves has been practised, but has rightly been discontinued. Suspension may be briefly described as follows:—a tripod and pulleys are provided similar to those used for the application of a Sayre's jacket; to the pulleys is attached an iron cross-bar; from the centre of this depends a leather apparatus, into which the patient's chin and occiput are fitted, and from the ends of it hang padded straps with which to support his axillæ. By the pulleys he is raised gradually off his feet, and suspended by head and arms, first for half a minute, and for longer periods on subsequent occasions till four minutes is reached. If thought advisable, he can be told to raise his arms now and then, so that he hangs by the head only. This process is repeated once a day for thirty days or more. Suspension, originally proposed by Motschutkowski, received the sanction of Charcot in France, and was introduced into this country, I believe, by Dr. De Watteville. Trustworthy observers have reported favourably upon this method, but it has scarcely fulfilled its early promise, and appears to be falling into disuse.

J. A. Ormerod.

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SPASTIC PARAPLEGIA

The present article deals only with Spastic Paraplegia and its modifications, and except in so far as may be necessary for the purposes of general clearness or of differential diagnosis, the other forms of paralysis attended by spastic symptoms which are described in other parts of this work are left out of consideration.

History.—Although it had long been recognised that some cases of paralysis are accompanied by rigidity and others are not, it is little more than twenty years since Spastic Paraplegia was definitely described. Professor Erb of Heidelberg gave the first clear account of it at the Wander-Sammlung of South-West German alienists at Heppenheim in May 1875.

In July 1875, and before we in this country had become acquainted with the communication of Erb, a case presented itself in my wards of the Royal Infirmary of Edinburgh, and I had the opportunity of showing it to the Editor of this work, and to many other leading members of the profession, who attended the meeting of the British Medical Association in Edinburgh in that year. In September 1875 an account of the case was published in the *Lancet*, under the title of "A new Form of Pseudo-Paraplegia." In the course of that paper it was pointed out that the peculiar features presented by the patient were "the almost perfect soundness of the sensory functions, and the undue excitability of the motor structures, whereby in the affected regions general contraction is induced either by attempts at voluntary movements or by peripheral irritation." After describing the features of the case, which precisely corresponded with those of spastic paraplegia as we now know it, the paper goes on to show that the clinical picture does not correspond to any form of disease at that time generally recognised. It argues with regard to its seat in the following words:—"What columns are affected? Certainly not the posterior; for the sensory functions are normal. Certainly not the gray matter to any considerable extent, for reflex action, electro-contractility, and the nutrition of the muscles are unimpaired. With equal certainty we may say that the antero-lateral columns are affected, as is proved by the abnormal motor-excitability. That the peculiar action is of spinal, not of cerebral origin, is proved by its occurrence in answer to irritation of the soles, as well as on voluntary effort." With regard to the nature of the lesion, it is argued that probably a degree of myelitis first existed, and that this was followed by a rigidity due to sclerosis. And as to treatment, it is said that "the object to be kept in view is the diminution of the excitability of the spinal cord. The application of ice-bags along the spine appears to be invariably followed by temporary improvement. When they have been applied for some time the patient bends his legs quite readily; though when he attempts to walk little difference is noticed. Ergot of rye has failed to do good; Conium has also failed; Chloral Hydrate appeared to prove in some degree beneficial, but latterly has been found to be of little use." It is clear that this case afforded an excellent example of the malady in a somewhat advanced stage; and it was a matter of regret that the patient left Edinburgh, and passed out of observation.

From 1875 onwards the disease has been well known. Erb's admirable and classical descriptions, which appeared in *Virchow's Archiv* for 1877, and in v. Ziemssen's *Cyclopædia of Practice of Medicine*, and again Charcot's able lectures have made the spinal forms familiar to the whole profession.

Various forms of spastic paralysis are now definitely recognised, namely:—

I. Simple Primary Spastic Paralysis, sometimes called Lateral Sclerosis, the typical form of the disease.

II. Primary Spastic Paralysis, complicated with symptoms indicative of other lesions of the cord; namely, (*a*) with muscular atrophy (Amyotrophic Lateral Sclerosis, Charcot's disease); (*b*) with ataxia (Combined Lateral and Posterior Sclerosis, Ataxic Paraplegia); (*c*) Pellagra; (*d*) Putnam and Dana's Combined Sclerosis of the lateral and posterior columns.

III. Spastic Paralysis, secondary to pre-existing lesions (*a*) of the cord, such as Potts' disease, with its attendant spinal changes, transverse myelitis, hæmorrhage into the cord, thrombosis, etc. (*b*) Secondary to intracranial disease: (i.) secondary to hemiplegia in the adult; (ii.) secondary to intracranial disease occurring during fœtal life, at the time of birth, or in early childhood—the group generally described as Cerebral Palsies of Infancy and Childhood, or Little's Disease, and presenting varieties in respect of date of origin, being pre-natal, natal, or post-natal, and, in respect of distribution, being hemiplegic, diplegic, paraplegic, or monoplegic. (This subject is dealt with in another part of this work.)

IV. Functional or Hysterical Spastic Paraplegia.

I. PRIMARY SPASTIC PARALYSIS (LATERAL SCLEROSIS)

Definition.—A chronic disease of the spinal cord, *consisting in* sclerosis of the descending fibres of the crossed pyramidal tracts: *characterised by* long-continued disease, with slow and insidious onset, marked at first by no sensory changes beyond a feeling of abnormal fatigue after even slight exertion, by no disturbance of motor functions, excepting slight rigidity of muscles and exaggeration of deep reflexes, by no change in the electrical reactions, or in the vaso-motor or trophic functions; at a later stage, when the disease is fully established, characterised still by freedom from sensory changes, but by much interference with movement, especially in locomotion; by the spastic condition of the muscles, which shows itself in rigidity, a peculiar gait, and an interference with voluntary movement, also by exaggerations of the knee-jerks and other deep reflexes, ankle clonus, knee clonus, resistance to sudden passive movement, and ultimately permanent contraction and shortening of the muscles; but as a rule—until a very advanced stage—unattended by changes in electrical reactions, or by vaso-motor or trophic changes, or other symptoms; *caused by* over-exertion, hereditary tendency, syphilis, and other conditions not yet ascertained; *resulting in* gradual disablement, due to the muscular rigidity, and ultimately in death from complications either proper to the nervous system or not.

Morbid anatomy and Pathology.—The pathology of this condition has given rise to much discussion and speculation, and it must be admitted that there is not yet enough satisfactory evidence to enable us to pronounce with absolute certainty upon its nature. Records of autopsies and of the microscopical examination of the diseased structures are surprisingly few in number,—a fact which is explained by the comparatively recent recognition of the disease, its long course, and the rarity of uncomplicated fatal cases.

The earliest observers, reasoning as we have seen from the clinical features of the malady, were agreed that the lesion must be situated in the lateral columns of the cord, and evidence is accumulating to prove the correctness of this view. For although the clinical features have been found associated with the lesions proper to multiple cerebro-spinal sclerosis, with tumours, with chronic myelitis, with changes in the anterior horns, with changes in other columns besides the lateral, with brain changes proper to general paralysis of the insane, and with syringomyelia, still there are some uncomplicated cases which have been held to indicate that the lesion is essentially, and may be solely, confined to the pyramidal tracts. A nearly pure case was investigated by Dr. Dreschfeld: the clinical history reported paralysis of the lower extremities, with marked contractions, increased tendon reflexes in both upper and lower extremities, absence of any sensory or trophic disturbance, of any bladder or rectal trouble, and of any brain symptoms—clinically a typical uncomplicated spastic paralysis. At the autopsy the brain and coverings were found normal, and the only disease obviousl presented was in the pyramidal tracts of the cord, where sclerosis wa found particularly in the dorsal region, becoming less marked in the cervic and lumbar regions. But the case was not quite pure, for there were sligl tokens of change in some parts of the gray matter. By the kindness, Dr. Dreschfeld I have carefully examined sections of the cord, and foun the changes in the gray matter quite distinct. Among the most convincin examples of the pure lesion as yet recorded are those of Minkowsk Strümpell, and Déjerine and Sottas. In most of the other cases examined the disease was less definitely limited than in these. In some instances other white tracts were involved; in some the gray matter. But even apart from autopsies of pure cases we are fully justified in believing, from a study of the phenomena of spasticity, and of the complicated forms, in the existence of a simple primary disease. As we know, a lesion certainly exists there in the form secondary to injuries of the upper part of the motor tract. In Charcot's disease we find the spastic condition explained by lesions in the pyramidal tracts associated with disease of the anterior horns, which accounts for the atrophy. In another form in which we find spasticity with incoördination, we have the explanation in a lesion of the lateral columns, combined with sclerosis of the posterior columns. In cases complicated with general paralysis of the insane we find the lesion in the lateral columns associated with cerebral changes. And therefore, when we meet with symptoms of spastic disease without other lesions, we seem entitled, until evidence to the contrary shall be forthcoming, to refer them to a lesion of the crossed pyramidal tracts.

The microscopic changes observed are similar to those in sclerosis elsewhere. There is an increase of neuroglia with numerous Deiter's cells, thickening of the walls of the arteries, with connective tissue proliferation round them. The nerve-fibres have disappeared in some places, in others are reduced to axis-cylinders.

According to Sir William Gowers, it is probable that the degeneration (which he thinks primarily affects the nerve-fibres themselves—the increase of connective tissue being secondary) begins in the termination of the fibres in the gray matter in the intra-cornual fibrillæ, and may even be limited to those terminal parts, so that the white columns might be found normal at death.

It has been generally maintained, since the condition was described, and the functions of the pyramidal tracts ascertained, that the lesion of the lateral columns produces the paresis by interfering with the transmission of impulses downwards from the cortical motor centres. This we are fully entitled to accept. But the difficulty is to explain the further or spastic phenomena. Various hypotheses have been suggested: it has been maintained that the lesion does not confine itself strictly to the pyramidal tracts, but spreads into the gray matter of the anterior cornua so as slightly to involve the motor cells and to irritate them. But this opinion is wholly unproved and highly improbable. There is no evidence that in the ordinary cases such extension of sclerosis occurs. In Dreschfeld's case the change in the anterior cornua was so slight that it does not seem to have induced the clinical phenomena proper to it. And, on the other hand, in those cases in which it does occur, it gives rise to the complex form of the disease, of which we have to speak later. Moreover, if such an extension did occur, its results could scarcely be those of mere irritation with exaggerative phenomena lasting through a period of years. In accordance with general laws it must pass on to a further stage of atrophic or other change.

Another hypothesis refers the phenomena to a cutting off of the inhibitory influences which proceed from cerebral centres. It originated in a research by Woroschiloff, conducted in Ludwig's laboratory, which proved that the lateral columns contain certain fibres which are inhibitory of reflex movements, and by the destruction of which reflex action becomes more pronounced and the muscular tonus increased. There is a good deal to be said in favour of this view. But this explanation is met by an insuperable objection, namely, that if mere breach of the tract would suffice to produce the spastic conditions, they ought to arise at once in every case of cerebral hæmorrhage or other acute disease which destroys the continuity of the motor fibres. This is not the case, as every-day experience proves. According to this explanation, moreover, we should expect that with all destructive lesions of these inhibitory fibres we ought to get at once an exaggeration of deep and of superficial reflexes, and at the same time an increase of the muscular tonus. Now these, although frequently, are not constantly affected together. The one set of reflexes may be found involved alone.

Dr. Hughlings Jackson has advanced another explanation which refers the changes to the action of the cerebellum. He contends that a powerful tonic influence proceeds from this organ, which in health is controlled to some extent by the influence of the cerebrum. But when this latter is cut off, the cerebellum, being uncontrolled, gives rise to the exaggerated phenomena. Yet, apart from the fact that such a function is by no means proved to belong to the cerebellum, we have here to face the same clinical argument which forms the chief difficulty in the way of accepting Woroschiloff's hypothesis; namely, that whenever the brain influence is cut off the exaggeration ought at once to become manifest: the spastic symptoms should not come on in the gradual way they do.

I am inclined to think that none of the hypotheses hitherto suggested affords a satisfactory explanation of the spastic phenomena. It is conceivable that they might result from changes in the efferent nerves, in the nerve-endings in the muscles, or in the muscles themselves; but I am not aware of any observations which lend support to any of these suggestions. The histology of these structures must be more minutely investigated, and it seems to me not improbable that new facts may be elicited of such a nature as to throw new light upon the spastic processes. One interesting observation was made some time ago by Brissaud, according to which the application of an Esmarch's tourniquet round a spastic limb, so as to render it bloodless, temporarily removed the spasticity; and this may be regarded as affording a certain measure of support to a hypothetical explanation of change in the muscles, or nerve-endings, or nerves.

But whether it should turn out that demonstrable histological alterations exist in these structures or not, we must bear in mind the possibility that spasticity may be due to molecular alterations in nerve-cells which are beyond the ken of the microscope, and that what we should be obliged to call undue functional activity in the anterior cornual cells may lie at the root of the process.

Clinical History.—The beginning of the disease is always insidious and its progress exceedingly slow. The earliest symptoms consist in a sense of fatigue on exertion, out of all proportion to the effort made, with scarcely any change in the gait, but with an exaggeration of the knee-jerks. The increase may be equal in the two legs, or the one may show it more distinctly than the other. When this condition has lasted for a varying time it usually passes into one which is unmistakable. It is quite conceivable that recovery may take place from this early stage; but I have never met with such a case; and it would always be difficult to find proof in any individual instance that the pre-existing symptoms had really been due to an incipient sclerosis.

When the malady is somewhat more advanced the features become unmistakable. The sense of fatigue is more easily induced than before, and walking is performed with effort. There is no difficulty in equilibration on standing; but when the patient walks all the muscles are in a state of extreme tension, like that which is instinctively assumed when walking on ice, or that which produces the peculiar strut of a Highland piper. In the production of this gait one element, no doubt, is the slight paresis, but much more important, especially in the earlier stages, is the high muscular tension and its associated heightened reflex irritability. When the patient lies down, especially after a long or rapid walk, he is apt to be troubled with clonic spasms of the feet and legs, the condition which Brown-Séquard used to describe as "spinal epilepsy." One sometimes sees this when there is no resistance to the foot, the patient being in the recumbent posture. Sometimes when the feet are on the ground, and the patient sitting, a paroxysm of clonic spasms is set up at the ankle, causing the foot to tap with great rapidity and the whole limb to vibrate.

On examination of the reflexes the knee-jerk is found greatly exaggerated; and often, in addition, one may notice that when, in answer to the tap over the ligamentum patellæ, the foot has been thrown forward, it is speedily checked as if firmly reined in, and the leg drawn back again by the unduly excited action of the flexors of the knee. If the patella be firmly grasped and suddenly thrust downwards, the limb being in the position of extension, a series of clonic contractions of the quadriceps extensor may sometimes be induced. This condition is known as knee clonus. Much more frequently we meet with ankle clonus. Sudden pressure upwards of the toes or the ball of the foot when the leg is placed in an easy, slightly flexed position, so as to produce a moderate degree of tension in the calf muscles, induces clonic contractions of the flexors and extensors of the ankle. It will be found that, while generally these movements are due to the gastrocnemius and soleus acting alternately with the antagonistic muscles on the front of the tibia, it sometimes happens that the peronei and the tibial muscles take the more prominent part.

If the disease be situated high enough in the spinal cord to involve the arms also, we may find exaggeration of muscular contraction when the tendon of the triceps, or of the biceps, or the bones at the elbow, or the tendons of the wrist are tapped. For the purpose of eliciting these deep reflexes a tap may be applied by the edge of the hand or by a thin book, but best by such a hammer as used for percussion, or by the earpiece of a stethoscope provided with a rim of india-rubber.

The skin reflexes are also markedly increased in some of these cases, but not in all; and in certain instances, although increased, they scarcely appear to be so, on account of the general rigid fixity of the limb which the attempt to elicit them induces.

In the earlier stages of the disease at least, gentle passive movements are possible without inducing spasm; but sudden passive movement induces powerful antagonistic muscular contractions such as to resist and suddenly to arrest it; or sometimes, while extending the limb, a spasm is set up, making the limb spring suddenly to a position of complete extension—the "clasp-knife" reaction, as it has been appropriately called. This is obviously the result of the undue irritability of the muscles.

The functions of the nervous system are otherwise unimpaired. All the sensory functions, the organic reflexes, the electrical reactions, and the vaso-motor and trophic functions remain normal.

At a somewhat later stage the condition already described is replaced by, or rather passes into one of greater rigidity and stiffness of the limbs. Movement is rendered difficult by the general spasmodic condition. The patient walks slowly and with effort; he is unable to lift his feet clear of the ground, and so drags the front part of the foot at each step. A glance at the patient's boots often shows that they are proportionally more worn down at the toes than elsewhere. This—the spastic gait—may occur occasionally when the patient is tired, even in the earlier strutting stage of the disease; but after a time it becomes constant. Erb explains its characteristic features—(*a*) by the paresis which causes dragging and catching of the toes; (*b*) by the muscular tension which causes stiffness of movement; and (*c*) by the heightened reflex action which explains the peculiar rise with each step. Spasm of the strong adductors of the thighs is often superadded, causing a closed position of the limbs greatly hindering progression, and inducing a tendency to the cross-legged gait, which, however, is more frequently exhibited by patients affected with the spastic cerebral paralysis of childhood. The various

DISEASES OF THE NERVOUS SYSTEM

(*CONTINUED*)

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DIFFUSE DISEASES OF SPINAL CORD

MYELITIS

By the name myelitis is meant inflammation of the spinal cord, but though pathologically the subject of inflammation could be reduced to moderate limits, the clinical results of myelitis in different cases are so varied, that the diseases which may be regarded as forms of myelitis make a considerable number. For in the nervous system the several functions have each a special localisation or anatomical seat, and thus the isolated disease of a particular anatomical position may lead to special and characteristic functional disturbances. Indeed, such a selection of parts of the spinal cord by an inflammatory process does take place; and this statement may still hold good, even should we have to admit that in some of the chronic forms of partial myelitis to which I refer a degenerative change sometimes occurs first of all in the nerve tubules, the lesion of the connective tissue being secondary. Thus, on the one hand, the fibrotic change may be confined to one tract of tissue such as the pyramidal tract, or portions of the anterior cornua, on the other it may occur throughout the whole thickness of the cord, involving white and gray matter indiscriminately, either in one continuous yet circumscribed area, or in separate small areas throughout the length of the cord. The diseases which result from the former condition were called by German writers System-Krankheiten, that is, diseases of systems, the systems being those parts of the spinal cord which are distinguished from one another, either by their period of embryonal development or by their function in the transmission of nerve impulses. In this work these diseases are described under their respective titles by other writers.

The present article will be almost confined to those forms of myelitis in which the systems or tracts are not specially represented, in which the lesions are not systematic, and which, in contradistinction to limited, may be called *diffuse*—by which qualifications must clearly be understood the kind of tissue involved, whether special tracts on the one hand or all the tissues of the cord on the other, and not the greater or less extent of the cord invaded as measured by inches.

By some writers these two varieties are spoken of as parenchymatous and interstitial, the "systematic" lesions being regarded as arising primarily in the nerve tissues themselves, the "diffuse" or "non-systematic" lesions as starting from changes in the vessels and connective tissues. Many of the former lesions, such as those of locomotor ataxy and Friedreich's ataxia, are also included under the term "sclerosis"; but this is a pathological condition which is also represented in a diffuse myelitis, at any rate in its later or chronic stages.

Confining ourselves now to the form of myelitis, and leaving aside the systematic diseases, we have to recognise that myelitis may be *acute* or *chronic*; and it will be convenient to deal with these two groups separately; even though we allow that many cases of so-called chronic myelitis began as acute attacks, of which they are only the uncured remains. In other cases, however, the disease begins slowly, and advances without any phase that can be called acute.

Other bases of classification may be adopted; for instance, the etiological: thus we may recognise forms of myelitis due to injury; to compression; to syphilis, and to other toxic or infectious causes. But in the nervous system the localisation of functions compels us to look to site as a basis for diagnosis; and consequently, even in the diffuse myelitis, both in its acute and chronic varieties, we may, by appropriate symptoms, distinguish the forms in which this or that part of the cord or the whole of the cord is concerned. Thus the lesion may be (i.) *widespread* or *disseminated*, or (ii.) *localised* in a small segment of the cord, and hence often called *transverse*. In the latter case the symptoms will be somewhat different, according as the lesion is the cervical, the mid-dorsal, or the lumbar region of the cord. These are the more common cases; but occasionally cases are observed in which the symptoms, or post-mortem evidence, show that the lesion is seated in (iii.) the central parts of the cord over a considerable vertical extent—a *central* myelitis, or in (iv.) the cortical or peripheral parts—a *peripheral* or *annular* myelitis, and (v.) lastly, perhaps most rarely, it may be limited, or almost limited, to one-half of the cord—a *unilateral* myelitis.

ACUTE MYELITIS

Etiology.—The causes of myelitis are not materially different from those of inflammation of the other organs of the body; and the disease is attributable, in different cases, to cold, to injury of various kinds, to the spread of contiguous inflammations; to the influence of infectious diseases, and to toxic conditions either associated with them, or arising in other, perhaps unknown ways.

Cold.—Pathologists are inclined to revolt against the notion of cold alone as a cause of local inflammation; though it has long held its place in the etiology of pneumonia, pleurisy, nephritis, and rheumatism. Nevertheless it is not infrequent in the history of paraplegia and myelitis to find that, shortly before the onset of symptoms, the patient had been exposed on a cold day: a navvy has been standing and digging in wet earth, or even in water; a cabman has been exposed for hours on the box. Often, indeed, the cold has been applied to the legs rather than to the spine, and, though the association of the cold legs and the subsequent paralysis is obvious to the patient, to the pathologist a link is still wanting to connect the depressed temperature of the legs with the inflammation which he knows is located in the spinal cord. Feinberg (4) produced disseminated myelitis by frequent applications of an ether spray to the shaven spine of rabbits; but these conditions are different from the more common circumstances under which myelitis occurs in man, and the experiments do not help us much to understand the details of the process; moreover, they were not confirmed by Roth (4). The modern belief in an almost universal bacteriological pathology would lead us to suppose that the cold acts by depressing the vitality of the spinal cord, and thus rendering it prone to succumb to bacteria or toxins.

Rosenthal and Thiroloix (23) report a case which they regard as demonstrating this connection. A man, aged 52, a polisher of parquet flooring, had suffered some fatigue in his legs from his occupation, and was exposed to chill by riding on an omnibus while still perspiring from his work. Within a few hours he had tingling in the toes, and the next morning he found his legs weak, and could not micturate properly. By mid-day he had complete paralysis of the limbs. He died four weeks later; and at the necropsy the cord was found soft and diffluent over an extent of 4-5 cm. in the mid-dorsal region. All the cultures for this focus gave a streptococcus identical with S. pyogenes, and when inoculated into a rabbit's ear it produced a characteristic erysipelas. A remarkable feature of the case is the rapid manifestation of the symptoms after the operation of the secondary factor—cold.

Injury.—This may occur in several ways; sometimes the patient has fallen on his feet from some height, and exposed his spine to shock or concussion; sometimes there has been a direct injury by a blow upon the spinal column; in other cases, again, there has been a strain or wrench of the spine. These are all acute changes, and it can be well understood how the spinal cord may suffer either by mechanical injury to its own tissues or by rupture or injury of its blood-vessels. It is not easy to see how a local injury should cause a widespread myelitis, as sometimes seems to happen. Still more remarkable is the fact that myelitis is rare out of all proportion to the number of instances in which accidents, apparently competent to produce such a lesion, occur; the additional factor determining the myelitis is not readily manifest.

Injury by compression is a frequent cause of local myelitis. The most familiar case is that of caries of the vertebræ, leading to the production of inflammatory materials, carious or suppurative, by which the spinal cord is compressed (see vol. vi. p. 851). New growths and aneurysms are also possible, but rarer causes of a similar lesion. In either case the compressed cord becomes the seat of inflammatory changes within a limited distance above and below the seat of pressure. In connection with injury may be mentioned divers' paralysis, or *caisson disease*, in which local injury and myelitis may be caused when the diver is submitted to sudden and considerable changes of atmospheric pressure (*vide* art. "Caisson Disease," p. 38).

Adjacent inflammation.—The spread of adjacent inflammations as a cause of myelitis is illustrated by its occurrence from the penetration of bedsores into the spinal canal; but the meninges are necessarily first of all involved; indeed a meningitis is at all times a possible source of irritation of the nerve tissue which lies within the membranes. The myelitis in such a case is often confined to the surface of the cord, and does not extend throughout its substance; it is then called *peripheral*, as opposed to a *central* myelitis, which occupies the axis of the structure. Another way in which the cord is secondarily inflamed is by the extension of a neuritis along the nerve-trunks and nerve-roots, a process which appears to arise in some cases. This is the explanation now generally adopted of the cases formerly described as **reflex paraplegia**, in which the symptoms of myelitis of the lower part of the spinal cord have arisen, as a sequel to diseases usually of the genito-urinary organs (cystitis, nephritis, prostatic disease), but also of the intestine, as in dysentery (vol. vi. p. 634).

Septic or toxic causes.—Of cases presumably due to toxic or septic agencies acting through the blood two groups may be made. In one the myelitis occurs as a sequel of a definite and well-recognised infectious disease; and the following are some of the diseases which are said to have had such a result:—small-pox, enteric fever, measles, diphtheria, influenza, puerperal fever, blennorrhagia. It is very properly remarked by Blocq that the records on which the above specification of diseases is based are not entirely trustworthy—in so far, at least, as they refer to cases noted before the general recognition of peripheral neuritis as a cause of pronounced paraplegia. The above diseases are equally apt to be followed by neuritis, and the relative frequency of myelitis and neuritis as sequels has yet to be estimated. With this group of diseases may be placed syphilis, to the paraplegic sequels of which attention has of late been much directed.

Mr. Hutchinson called attention to the frequent occurrence in the earlier periods of syphilitic infection—that is, up to two years from the date of infection—of different kinds of nervous disease, and especially of a form of acute paraplegia, which presented all or many of the characteristics of a myelitis. His observations are confirmed by many neurologists at home and abroad. He noted that, though most of these patients improved, almost none got perfectly well, and a few died. In some of the fatal cases a definite myelitis was found; but it is clear that the proof of the syphilitic nature in these cases rests only upon the antecedent occurrence of syphilitic infection, and is confirmed neither by any specific anatomical appearances nor very definitely by the results of treatment.

For the present, then, it is sufficient to say that it is allowed that within from six to twenty-four months after the primary affection, a condition of paraplegia may arise which is due probably to a true myelitis, and certainly not to the more circumscribed and usually much later lesion, the syphilitic gumma.

To the second group belongs a number of cases in which, without any special antecedent (unless, perhaps, such doubtful disturbances as cold and strain may sometimes be), the wide dissemination of the lesion suggests a septic or toxic, widespread, blood-borne origin. As might be expected, the subject of "infectious myelitis" has created a good deal of interest in recent years; and the possibility of its occurrence has been shown by various researches not only clinical and pathological, but also bacteriological and experimental. H. Claude (9) injected into guinea-pigs the filtered bouillon of strepto-staphylococci: many of the animals were unaffected; but two of them showed paralytic symptoms after a fortnight, and died within two or three days. The spinal cords were injected, and presented minute hæmorrhagic foci; the small vessels were dilated, and their sheaths were filled with leucocytes. Claude also caused a subacute paraplegia in a dog by the injection of small doses of tetanic toxin over a period of twenty-four days. The dog was killed about six weeks later, when the spinal cord was found to present numerous centres of disseminated myelitis, especially in the cervical and lumbar enlargements. Babes and Vassali record cases of paraplegia in man which they regard as showing the infective origin of a myelitis. In one case the micro-organism of malignant œdema was found in other organs; in another, a short streptococcus was obtained from the vessels, and some vessels of the anterior cornua were filled with streptococcus thrombi. These authors, as well as others, regard the existence of early changes in the small blood-vessels (thickening of their walls, distension of the sheaths with round cells) as evidence that the source of the disease was in the vessels, and must therefore be a toxin or other virus conveyed thereby. Rosenthal and Thiroloix (24), in a case of vegetative streptococcal endocarditis, in which an acute paraplegia appeared in the last three days, found that the spinal cord was extensively softened in the dorsal region; and a culture of streptococcus was obtained from the lesion. Their other case of streptococcal myelitis has already been mentioned (23).

Drs. Buzzard and Risien Russell read before the Clinical Society a case of acute ascending meningo-myelitis which was fatal in thirteen days. The cord was engorged and softened, and had numerous extravasations of blood. A diplococcus was found in the meningeal exudation, and in the substance of the cord; and from the former situation pure cultures were obtained. Dr. Russell also stated that he had since examined a case of acute myelitis, without meningitis, in which the same organism was obtained.

As previously stated, the occurrence of numerous small foci of disease throughout the cord, a so-called disseminated myelitis, has been regarded as proof of a septic origin; and this seems all the more probable when the myelitis occurs with inflammation of some other and simultaneously remote part of the nervous system such as the optic nerve. Several cases of the association of myelitis and optic neuritis have now been recorded; and they seem explicable only on some such view as this. But after all it would be very remarkable if the spinal cord were not in some degree, at least, subject to the same septic processes as other organs; and the cases above recorded are probably the forerunners of many others proving the relations of myelitis to sepsis.

Pathological anatomy.—*Anatomically* myelitis results in changes which are analogous to those occurring in other organs the subject of inflammation; namely, increased vascularity, extravasation of corpuscles from the blood-vessels, certain changes in the specialised structures tending to destruction, and, later, the replacement of those structures by an increase of the connective tissues.

When removed from the spinal canal, the cord, from the greater tension of its containing membrane, may seem to be hard; but on section it is often found to be extremely soft, the substance of the cord becoming everted over the marginal pia mater, or almost flowing out like cream. On the other hand, it may sometimes show no material alteration in consistency either before or after section. In colour it is sometimes redder than normal, especially in the central gray matter; often there is no increase of vascularity, but it is difficult to recognise the outline of the gray matter; its margin is blurred and indistinct, or some areas of the white matter present a gray tint. However, the colour of the cord is likely to vary with the stage in which death has occurred; it is likely to be redder in early stages, more yellow later, and more gray after this. Sometimes nothing definite can be said about the cord until a microscopic examination has been made. And it may here be noted that the more softened the cord, the more difficult it is to get satisfactory results from the usual hardening processes; it is therefore generally desirable to examine some portion of the cord under the microscope in the fresh condition, when some of the changes at least may be observed sufficiently well.

The following are the histological changes seen in acute myelitis:—The *vessels* are dilated and distended with blood, and their lymphatic sheaths are filled with red blood corpuscles or leucocytes, or granular material, or large cells which may be proliferating endothelial cells. Sometimes minute hæmorrhages take place from the congested vessels. The *nerve elements* first become swollen, and afterwards degenerate or break up. The *nerve cells* may become enormously enlarged, according to Charcot, up to a diameter of $\frac{1}{30}$th inch (·825 mm.); the protoplasm becomes granular and the nuclei disappear, or vacuolation occurs, or colloid degeneration. The prolongations are thickened or disappear; and the cells become rounded and deformed, and ultimately themselves atrophy completely. The nerve-fibres undergo changes both in the axis-cylinders and the myelin. The axis-cylinders are subject to a curious process of enlargement (so-called hypertrophy), in the form of elongated fusiform swellings, by which the diameter of the axis-cylinder is increased to four or five times the normal. These swollen fibres occur in scattered groups, especially in the white matter. The myelin of the fibres soon becomes disintegrated, breaks up into short segments, and appears as minute drops or globules among the debris of the nerve tissue.

The *connective tissue* of the cord shares in the inflammation by a proliferation of its elements. It is infiltrated with nuclei and red blood corpuscles; its cells are enlarged, and especially the cells known as Deiter's cells become very distinct and numerous. A comparatively early product of inflammation of the spinal cord, as of that of the brain, is the granule-corpuscle of Gluge, a large globular body containing closely-packed, opaque, fat globules, and arising probably from the proliferating neuroglia cells and droplets of disintegrated myelin. These changes occur quite early; for instance, hypertrophied axis-cylinders and abundant Gluge's corpuscles were present in a case fatal on the eleventh day from the first symptom. In cases of longer standing the changes which take place are in the direction of increased connective tissue and disappearance of the special nervous elements. Granular corpuscles are still seen months afterwards. The neuroglia gradually assumes the appearance of a dense but finely-fibred felt-work, with scattered nuclei.

A true abscess of the spinal cord is a rare event, and is probably always associated with a purulent meningitis.

I must not omit to say that for some writers, for Dr. Bastian, for example (2), a true acute myelitis is of the rarest occurrence; and that the majority of the changes described under this head are only conditions of softening dependent upon loss of nutrition from obstruction of vessels. It appears to me that the more recent researches into different forms of infectious myelitis are strongly opposed to such an interpretation of the facts.

As already stated, the positions which the lesions of myelitis occupy in the spinal cord are variable, and give rise to clinical groups of symptoms which may be distinctive. In the first place, the changes may be limited to one or more segments of the cord corresponding to one, two, or three vertebræ, and affecting the cord more or less completely to that extent. This is called a *localised* or *transverse myelitis*. It may be determined by disease in the adjacent vertebræ, as in compression myelitis, from caries of the spine, or by an abscess lying near the spine, or other local disease (vol. vi. p. 854); or, on the other hand, it may arise from causes not manifest either in the history or after death. In a second group of cases the cord is affected either throughout, from end to end, or over a greater or less vertical extent; but in any case greater than would conform to the word transverse. Such a lesion might fairly be called diffuse, in distinction to localised, but that we have employed the word in another sense. As a matter of fact, even if the cord is to the touch or to the naked eye softened throughout, it will generally be found on hardening and staining that there is still a quantity of healthy cord tissue, and that the lesions are numerous, small, and scattered or disseminated. So that the term *disseminate* may be used to distinguish this group. These two varieties form the majority of the cases. The transverse or localised forms may, of course, be divided further, according as the lesion is in the cervical, dorsal, or lumbar region; and the less completely disseminated cases may occupy in some instances chiefly the upper, in others chiefly the lower part of the cord. A *central* myelitis is spoken of by many authors: its meaning seems plain, but the term is used with somewhat various significations. In other cases the surface of the cord is inflamed, so that on section a narrow ring at the periphery of the section is seen to be affected. This may arise as a consequence or as a part of spinal meningitis, and is called, as I have said, *annular* or *peripheral myelitis*.

Secondary degeneration.—A constant result of the persistence of myelitis when limited to any small extent of the spinal cord is the occurrence of secondary degenerations. These consist of a subacute or chronic change in the originally healthy white columns above and below the lesion; and the columns habitually and chiefly affected are the posterior median columns above, and the posterior part of the lateral columns, corresponding to the pyramidal tracts, below. Thus the fibres in which impulses are habitually transmitted from the seat of the lesion to either extremity of the cord are affected, and not those in which impulses are transmitted towards it; or, more correctly, those fibres degenerate which are cut off from their trophic centres, or of which the trophic centres are destroyed by the inflammatory lesion. The degeneration of the pyramidal tracts below the lesion is of the same nature, and has the same explanation, as that which occurs in the pyramidal tract of one side as the result of a cerebral lesion involving the internal capsule, and producing hemiplegia. The degeneration of Goll's columns is open to a similar explanation. In the one case the trophic centre is above, and degeneration takes place downwards; in the other case the trophic centre is below, and the fibres degenerate upwards.

The degenerative process results in a sclerosis or hardening of the tract involved, and is in part atrophic, in part inflammatory. The axis-cylinder of the nerve-fibres is first affected by degeneration, the myeline is next involved, and finally the neuroglia undergoes inflammatory changes; thus thickening and sclerosis result, and the new tissue replaces more or less the atrophied nerve element.

Descending degeneration.—In localised diseases of the spinal cord, such as a transverse myelitis, the tracts affected by descending sclerosis (below the lesion) are (i.) the direct pyramidal tracts on the inner side of the anterior column, adjacent to the anterior fissure; (ii.) the crossed pyramidal tract, situate in the posterior part of the antero-lateral column; and (iii.), sometimes, the small comma tract of Schultze in the middle of the postero-external tract or column of Burdach.

Ascending degeneration.—In similar diseases of the spinal cord, the tracts affected by ascending sclerosis (above the lesion) are (i.) the posterior columns, especially the posterior median column, or column of Goll; (ii.) the cerebellar tract, and, occasionally, (iii.) the antero-lateral ascending tract, or tract of Gowers. For a short distance above the lesion there is degeneration of all the ascending fibres—above this the postero-external column of Burdach is involved to a small extent; but from this point the postero-median columns or columns of Goll are affected, and the sclerosis can be traced up to the bulb; the higher the situation of the lesion, the more complete is the degeneration of these columns.

Symptoms.—The symptoms may be divided into the purely local and the more general. The local symptoms, those, that is, that depend on disturbance of the functions of the spinal cord, are the most important.

Now the cord has to be considered as in part a series of nerve-centres, and in part a medium of transmission of impulses to and from the brain; hence the functions affected may be those to which it contributes by its conducting powers, or those which have their seat in its own structures. In so far as it is a conductor of impulses, its lesion will result in loss or impairment of voluntary movements in the parts below the lesion, in loss or modification of the various kinds of sensation, and in loss or modification of control of the bladder and rectum. On the other hand, the lesion of the nerve-centres or nerve-roots in the cord will result in some or all of the following changes in the parts immediately connected with these centres or roots:—loss or impairment of movement; pain or loss or modification of sensation; loss or modification of trophic influence in the muscles, skin, or bones; loss or weakening of reflexes, cutaneous and deep.

As the conduction of the cord is chiefly in a longitudinal direction from above downwards, this function will be affected by any transverse lesion at an isolated spot; such as a crush, transverse division by a wound, localised pressure by tumour or inflammatory products, or a local inflammation or hæmorrhage. Whether the functions of the cord as a central organ are much or little affected by such lesions will depend upon the vertical extent of the lesion; and in any case they will be less obvious when the lesion is in the dorsal region, of which the centres control mainly the spinal, intercostal, and abdominal muscles, than when it is in the cervical or lumbar enlargement; in this latter case the more specialised muscles of the upper or of the lower limbs, and the operations of the bladder and the rectum may be involved.

But an important observation has to be made with regard to those nerve-centres of the cord which are situated below the lesion whenever the lesion is far enough above the lower end of the cord to spare some if not all of the lumbar enlargement; or even dorsal centres above these. If they are spared, the functions of nutrition of the limbs or corresponding parts of the body will remain intact, and the normal electrical reactions will be preserved. The limbs, though useless, and more or less completely insensitive, will be well nourished, and will contract to galvanic and faradic stimuli. Reflex action, however, another function of these untouched centres, is not only preserved, but, as a rule, exaggerated. This exaggeration is obvious in an increase of the knee-jerk, in the occurrence of ankle clonus, and in the behaviour of the bladder in certain cases, when the accumulation even of a small amount of urine in the bladder is sufficient to set up reflex contraction of this organ, without the volition or even the consciousness of the patient.

As a late result of myelitis, and nearly always in association with these conditions of increased knee-jerk and ankle clonus, the muscles are often the subject of a peculiar kind of rigidity or persistent involuntary contraction. This contraction was regarded by Charcot as arising from an irritation set up in the motor cells of the anterior cornua by the spread of inflammation from the secondarily degenerated pyramidal tracts. Others, with more probability, as it seems to me, regard it as a reflex phenomenon probably due, with the ankle clonus and the heightened phase of the knee-jerk, to the loss of cerebral control or inhibition, of which the lower centres are deprived by the lesion cutting transversely across the line of conduction of impulses from the brain.

These reflexes are discussed in a special article (vol. vi. p. 528), but I must at least point out that it is now admitted that under certain circumstances a transverse lesion of the cord, leaving the parts below, the lumbar enlargement, for example, absolutely intact, may be accompanied by perfect flaccidity of the muscles, and an abolition, instead of the maintenance and exaggeration, of the various reflexes. The necessary condition appears to be that the lesion shall be a complete lesion, such as will totally intercept impulses whether of motion or sensation between the brain and the extremities, and will there cause complete paralysis and complete anæsthesia. The existence of such cases has been demonstrated, for spinal softening and compression, by Dr. Bastian (3), and for severe traumatic lesions by Mr. Bowlby and other surgeons; and, although the explanations given so far can scarcely be said to be satisfactory, of the fact itself there can be no doubt. Hence we are in this position, that the persistence or increase of the knee-jerks of the lower extremities implies the integrity of certain lumbar centres; but that the loss of these reflexes may be brought about either by a destruction of these lumbar centres, or by a complete transverse lesion above these centres, while they themselves are intact.

In a disseminated or widespread lesion the functions of the cord will be more or less completely abolished from the upper limit of the lesion downwards. Thus, if the lesion extend sufficiently into the cervical enlargement, there is paralysis of all the muscles of the legs and trunk, and of the arms, anæsthesia up to the same level, paralysis of the sphincters of the bladder and rectum, complete flaccidity of muscles, loss of reflexes, loss of electrical reactions, and, if the case last sufficiently long, wasting of the muscles.

ACUTE DISSEMINATED MYELITIS.—In the detailed description of the symptoms and clinical features of acute myelitis, it will be convenient to take, first of all, the condition of acute disseminated myelitis. The short notes of a case will illustrate at least one group:—

A man, aged 32, slipped while getting out of a train, and received a slight shock, of which he took but little notice. He did not fall to the ground, but he remained on his legs, and his body was bent backwards. Suddenly, perhaps to keep his equilibrium, it then bent forwards, and he felt a little pain in the loins. After the drop he shivered and felt pains in his body. He continued his work the two following days. The next day was the first on which a spinal symptom was noted. He attended a funeral, walking a distance of ten miles; here he got wet, and at the end of the journey was unable to hold his water. The next (the second) day he walked the same distance back, complaining of weakness in the legs, and reaching home with difficulty. On the following (or third) day he had to have his urine drawn off, and he felt numbness in his legs. The fourth day he got up with difficulty, and was laid up completely after this. On the seventh day he passed motions involuntarily in bed, and on this and the following day he had temperatures of 102° and 103°. On the ninth day, now being in hospital, he was completely paralysed in the lower extremities, and was able to move only the head and the hands, but the hands were weak—the right more than the left; the lower part of the chest was immovable, and the breathing was diaphragmatic. The voice was clear, but he had a short weak cough and could not expectorate. The abdomen was tumid, tympanitic, not resistant. He had completely lost sensation up to the level of the seventh rib on both sides; there was a sensation of pins and needles in the upper extremities, but none in the lower. There was no reflex action in the legs (this was before the use of the knee-jerks as a test), and the muscles were not rigid. The electrical reactions, both galvanic and faradic currents, were normal. Râles were heard in the chest, and air entered the posterior bases badly. The temperature on this and the next two days continued to be high, between 101·5° and 104°. On the eleventh day he had almost entirely lost power in the right hand, the thoracic muscles were completely paralysed, the face was livid, and the mind occasionally wandered. At night he was delirious. The urine, drawn off by a catheter, was bloody and strongly ammoniacal. On the twelfth day the dyspnœa was extreme, he spoke with difficulty, but his mind was clear, and he could still move his left hand. He died at mid-day.

The necropsy showed that there was extensive softening of the spinal cord from a point three inches above the lower extremity to a point nine inches higher up; the softening being in disseminated small patches, irregular in size and shape.

Thus the characteristic feature of this condition is a rapidly ascending paralysis, beginning either with numbness and tingling, or with weakness of the legs, or with some disturbance of the function of the bladder. The paralysis becomes complete, sensory functions are abolished, and both paralysis and anæsthesia extend so to involve the abdomen, and perhaps the lower part of the chest; the upper limit of the anæsthesia is at first over the abdomen, and reaches later to the lower sternum or nipple level. The reflexes are abolished in the lower extremities; the bladder is paralysed; and retention of urine occurs, so as to require the use of the catheter. The fæces are discharged involuntarily and unconsciously. As the mischief extends upwards the intercostal muscles are more or less paralysed; the circulation in and movement of the bases of the lungs are impaired; mucus accumulates in the bronchial tubes, cough and expectoration are increasingly difficult; and in this way death may be rapidly brought about; as in the above case, which lasted only twelve days. In other cases the symptoms may be prolonged for three or four or six weeks; or still longer, if the respiratory muscles are spared.

Confining ourselves to the more acute cases, the temperature is usually high, and there may be some delirium at night, especially towards the end when respiration is failing. Unless extreme care be taken in the use of catheters, cystitis will be rapidly induced, and may take an intense form, with extravasation of blood into the urine and into the mucous membrane of the bladder. The early tendency to bedsore is another way in which the loss of nutritive power is manifested in the tissues of the paralysed parts. In the early days of the illness the electrical reactions of the muscles may not be much altered; but probably after a week or ten days the faradic current will fail to elicit normal contractions, and later it will cause no contractions at all; at the same time the galvanic current will give the modified results characteristic of the reaction of degeneration (A.C.C. = or > K.C.C.) Similarly the muscles, though completely flaccid, will preserve their bulk for some days, and in the rapidly fatal cases show but little difference from the normal.

In the event of the patient not being carried off by the rapid implication of the respiratory functions, life may be maintained for several months, when the case passes from the acute condition to one of chronic persistence of damaged tissues, rather than of chronic myelitis.

The symptoms remain practically unchanged, or vary a little from week to week. Some power in the movement of a toe or the slight contraction of a muscle may be regained; the patient may allow that he has one day a little feeling in a part that was perfectly anæsthetic; or there is a slight variation in the height to which the anæsthesia can be traced; or the superficial reflexes may appear and disappear. But the muscles become wasted and flaccid; the patient is quite helpless in the lower extremities; and the faradic and galvanic reactions are more or less completely lost. So far the patient, though incapable, might seem to run no risk of his life; but danger lies in the tendency to cystitis and to bedsores, both of which are avoided with difficulty, and either of them may be a cause of death—the cystitis by leading to consecutive (suppurative) nephritis and uræmia, the bedsores by septic absorption.

Recovery in these cases does, however, sometimes take place; the symptoms gradually subside, and the limbs recover power and sensation.

While the above represents the essentials of a case of disseminated myelitis some occasional features may be mentioned. In regard to sensory functions analgesia accompanies anæsthesia; and at the beginning the subjective sensations may include, besides a sense of numbness, the sense of tingling, pricking, or formication. In some cases the upper limit of the area of anæsthesia is bounded by a band of skin, varying from one to five or six inches in breadth, in which hyperæsthesia is present; and at the upper limit of this the skin is normally sensitive. This hyperæsthetic band may pass transversely round the entire trunk, and almost perfectly horizontally, little if at all inclined in the direction of the course of the intercostal nerve. At the same time slight undulations in the track of the upper and lower limits may be observed.

The bladder is paralysed, and the patient at first suffers from retention of urine, so that the bladder may become distended up to the level of the umbilicus; and in one such case, in a woman, 56 ounces of urine were drawn off by the catheter. If the distended bladder be left unrelieved, the sphincter yields, and the excess of urine comes away, forming the so-called "overflow incontinence"; but in any patient coming under treatment the regular use of the catheter is necessarily adopted, and the consequences of excessive distension are avoided. But one result of the inability of the bladder to expel the urine by its own efforts is very difficult to avoid, namely, the supervention of cystitis, which may be most intense. No doubt it is due to the entrance of micro-organisms, which may be conveyed in the catheters, introduced as they are usually at least twice daily, but conceivably in some cases they pass along the urethra without such assistance. The urine becomes ammoniacal, turbid, even bloody; and deposits crystals of ammonio-magnesian phosphates, leucocytes, pus-cells, bladder epithelium, and blood corpuscles.

Bedsores, no doubt, are determined by the pressure which is exerted by the weight of the body upon the parts immediately in contact with the bed, and by the decomposition of discharges which soil the skin; thus the more complete the paralysis and anæsthesia the more likely is the patient to have bedsores. The patient neither feels the discomfort of the prolonged pressure, nor, if he felt it, could he shift his position to relieve the suffering part. The pressure deprives the part of its blood, and hence its nutrition is affected and the part is apt to slough. If urinary or fæcal soiling of the skin be permitted a bedsore will soon appear; on the other hand, scrupulous cleanliness and antisepsis of the skin, with frequent changes of the patient's posture, will prevent this distressing complication.

Sir William Gowers records some cases of rapidly increasing ataxy, the dependence of which upon an organic spinal lesion seemed certain, while their onset was incompatible with any other than an inflammatory process. Hence he regards them as cases of *acute myelitic ataxy*. One case was that of a woman, in whom, twelve months after the occurrence of secondary symptoms of syphilis, complete incoördination of the legs came on in thirty-six hours. The knee-jerk was normal on the right side, almost lost on the left; the left leg was thought to be weak at first, but its power was found to be good in a day or two. With this was hyperæsthesia of the lower part of the trunk and legs, soon reduced to a band round the lower abdomen; later, there was a girdle pain in the same situation. She could walk a little, though still highly ataxic, in four months; and ultimately recovered. In other cases Gowers saw incoördination of one arm, with complete muscular anæsthesia, arise in the course of a few hours, and only slowly subside.

ACUTE DISSEMINATED ENCEPHALOMYELITIS.—A form of disseminated myelitis, combined with a similar lesion of the brain, is described under this name (7); numerous small foci of inflammation are found in the cord, in the pons, and in the cerebrum. It is called *acute central ataxia* by von Leyden.

The disease nearly always occurs during or soon after an infectious disease or similar process; consequently a large proportion of the cases are in children. The onset is sudden and suggests the implication of the whole central nervous system. There is often unconsciousness, either with paralysis or with restlessness and delusion; and after a time the unconsciousness clears up, and a stage is reached which is characterised by ataxia or choreic movements. In other cases the sudden cerebral symptoms are absent, and ataxia is the first symptom. In this stage there are ataxic movements or intention-tremors of the legs, arms, and head; disorders of speech, especially of the scanning variety, but sometimes dysarthric; and sometimes disturbances of intellect. The gait is variable; it may be ataxic, or spastic, or paretic, or tremulous. The arms are less commonly ataxic, but more often present intention-tremor; and the same occurs with the head. Nystagmus appears to be uncommon. There is great variety in the subsequent course: a few cases recover quickly and permanently; many end in recovery after months, or after one or two relapses; in other cases, some symptoms, especially speech disturbances, persist; but death is rare. Aphasia or optic neuritis occasionally occurs in the earliest stage. The resemblance in many points to disseminated sclerosis is obvious, and it might be thought that if the disease persisted, the pathological conditions and clinical symptoms of multiple sclerosis would be ultimately assumed. German observers state, however, that this has never been observed.

The association of *optic neuritis* with myelitis has been observed on several occasions, and presents interesting features. Bibliographies up to date are given by Devic and Katz; and I have recently published a fatal case myself, though unfortunately without necropsy (26). In a large proportion of the cases (15 out of 21 collected by Katz) the ocular affection precedes the spinal lesion by a period varying from a few days to some weeks; in nearly all the remainder it occurs simultaneously. There is generally a rapidly advancing amaurosis in one or both eyes, and if in both simultaneously, in one eye before the other. Generally the optic disc shows swelling and hyperæmia; but the changes in the optic nerve may be situated behind the eyeball, and the papilla may be unaffected until after some weeks it becomes atrophied. The myelitis is commonly situated in the dorso-lumbar region, and is manifested by anæsthesia and paralysis of the lower extremities, with extension for a variable distance up the trunk, and paralysis of the bladder and rectum. Indeed it differs in no respect from a rather widespread or disseminated myelitis. A good many cases are reported to have ended in recovery; so that the prognosis is better than the wide extent of the disease would lead one to anticipate. Its pathology may be still open to doubt, but it seems likely that some common infection is the cause of the inflammatory lesions in parts so remote from each other as the optic nerve and the dorsal portion of the cord. The suggestion of an ascending meningitis has not been confirmed by necropsy. For the most part disseminated foci of acute myelitis have been found irregularly distributed in different parts of the cord; the vessels over-filled with blood, the perivascular spaces crowded with round cells, leucocytes, granule-corpuscles, and debris.

LOCAL MYELITIS.—**Transverse myelitis.**—This is common in the dorsal region; and it will be convenient to describe this variety first, because it concerns a part of the cord with the least specialised functions, and then to describe the myelitis seated in the cervical and lumbar regions.

If the myelitis be due to local disease in the neighbourhood, such as caries or tumour of the spine, the symptoms indicative of the implication of the cord may be preceded by those due to the primary disease, of which pain is likely to be the most important. But even in other cases local pain may sometimes be an early sign, occurring in the part of the back which corresponds to the seat of the myelitis. Then more or less rapidly occur the conditions which are due to the disease of the cord; namely, either numbness, tingling, or formication in the lower extremities, or weakness of one or both legs, or inability to pass urine. Perhaps the sensory failure is the more common mode of onset. Thereupon, with varying degrees of rapidity, these three conditions become aggravated; the paralysis and anæsthesia involve the legs, thighs, and more or less of the abdomen, the upward extent being determined, of course, by the position of the lesion in the cord, or rather by the upper limit of the lesion. Supposing, for instance, the lesion involves but reaches no higher than the segment from which the seventh dorsal nerve comes off, all the muscles supplied by the last six dorsal nerves, the lumbar and the sacral nerves will be paralysed; and anæsthesia will be found in the area of the body corresponding to the same nerve-roots.

The loss of power is more or less complete according to the thoroughness of the lesion, and the completeness with which the transmission of motor impulses is interfered with; but there is no other recognisable alteration in motor power as such: and neither tremors, nor spasms, nor convulsions occur, as a result of myelitis, in the muscles supplied by nerves connected with inflamed segments.

With a severe lesion sensation of touch, pain and temperature is completely lost; but with less complete damage some form of sensation may remain; analgesia, for instance, may be marked when anæsthesia is incomplete, and the sense of temperature may be imperfect. Subjective sensations of numbness and tingling are less complained of after the first onset of the disease; but a sensation of pain at the upper limit of the anæsthesia, encircling the body, is often present, and is commonly known as girdle-pain. The band of hyperæsthesia described in the cases of disseminated myelitis may also be present in these more circumscribed cases.

The reflexes, both cutaneous and deep, are, as a rule, not only present, but increased in the parts of the body below the lesion. Of the deep reflexes there are two forms. One is the knee-jerk, which is seen to be exaggerated when tested in the usual way by a blow below the patella; it is also easily elicited by striking above the patella: the other is the ankle clonus, which, absent in health, is easily brought out in these cases by sharp flexion of the ankle-joint. In some cases the excitability of the lumbar spinal centres is such that the attempt to get the knee-jerk produces a more or less continuous clonus at the knee-joint, of the same kind as ankle clonus, but persisting, without such further handling of the part as is required in ankle clonus.

The exception to the rule that the reflexes are increased in the case of a limited transverse lesion above the centres from the legs is the case in which the lesion is absolutely complete throughout the whole thickness of the cord, as already mentioned (p. 12). In such a case the reflexes are absent; the muscles are quite flaccid, and the anæsthesia is complete.

The nutrition of the muscles of the part paralysed—that is, the lower limbs—is not materially affected; they lose a little of this from the fact that they are not used so much as in health; but they do not show the pronounced atrophy which results from lesions of the anterior cornua or nerve-trunks. The muscles are, indeed, in direct connection with undamaged spinal centres; and whether the nutrition be dependent on "trophic" centres, or simply, as Dr. Poore thinks, on repeated stimuli emanating from the spinal nuclei, the condition is unaffected by a lesion situated above the anterior cornual cells with which the muscles concerned are associated; in other words, the lower motor neurons are intact. Similarly, the electrical reactions are normal; the muscles react to faradism and to galvanism, and the galvanic reactions present the normal relation of kathodal to anodal closure, that is, K.C.C. > A.C.C. Another phenomenon, which is closely related to the increase of the reflexes, may also present itself in the later stages of the acute cases, and constantly in those cases which, beginning acutely, remain uncured, and hence pass into the group of chronic myelitis; this is *rigidity* or *spastic contraction* of the muscles. Like the clonus or exaggerated knee-jerk, it is always associated with an undamaged reflex apparatus, and with the separation of the spinal centres from the central motor area by some lesion or other, that is, a lesion affecting the upper motor neuron. In the absence of cerebral control it seems likely that constant stimuli from the cutaneous or other afferent nerves are able to keep up a constant slight motor contraction. However, the rigidity of the muscles under these circumstances is a very variable condition. In slight cases it may easily be overlooked. The limbs may lie flaccid in bed, and if the paralysis is imperfect the patient may be able to move the limbs slightly; but if the limb be handled by the observer, and flexed or extended at the knee or hip, it will soon be noticed that the passive movements at first easy become more difficult, and the leg gets so stiff that it can no longer perhaps be bent or straightened at all. If the legs are then left untouched the muscles quickly relax. In other cases the legs are constantly stiff, without any external interference; or if not actually rigid on being first observed, they become so directly they are touched, or directly the patient makes an attempt to move them. The legs may be rigidly extended, the muscles in firm contraction and closely adducted, so that the attempt to pull one leg to its outer side brings the other leg along with it. In other cases this condition of rigidity is associated with involuntary reflex contractions of the flexor muscles; and in such cases the patients often find when they wake up that the legs are flexed with the knees on the abdomen, and the heels near the buttocks. Sometimes the flexion relaxes, but the patients often have to assist the process of extension by the use of their hands.

The functions of the bladder and rectum are affected for the most part in a similar way. The centres for these organs are seated in the lowest part of the lumbar enlargement. An acute lesion, such as myelitis often is, though situate in the dorsal region, commonly causes at first paralysis of the detrusor urinæ, and consequently retention of urine. After a time the reflex becomes again established, and the bladder involuntarily contracts as soon as the urine accumulates to a certain extent, and thereby stimulates the walls of the bladder (reflex incontinence). If the transverse spinal lesion be complete, the desire to micturate will be lost, and the act of micturition will be unperceived, because the afferent fibres from the bladder are cut across; on the other hand, the interruption of efferent fibres prevents the process of micturition being either hastened or retarded by any effort of the will. In less complete lesions either of these functions may be imperfectly represented.

In the case of the rectum, paralysis with resulting constipation frequently accompanies a lesion in the dorsal region. A "reflex incontinence" of fæces may also occur, but is less common.

The general condition of a patient with a transverse myelitis varies with the stage of the disease. In the early days of an acute myelitis there may be a slight degree of fever, with the usual accompaniments; but, if it be not rapidly fatal, the patient will soon arrive at a stage in which the symptoms above described, and directly referable to the interruption of communication through the dorsal cord, are the only troubles. However, in any case of acute myelitis we have to consider the possibility of two complications arising, namely, cystitis and bedsore. The former has already been referred to under disseminated myelitis; it is much more likely to arise in the period of retention; if this stage be safely passed and a condition of "reflex incontinence" be present, cystitis may be avoided and the bladder may remain healthy.

The liability to bedsores in dorsal myelitis is variable; they are more likely to occur where the lesion is extensive, involving motion and sensation very completely, and where the nursing and care of the patient are inadequate. They are less prone to occur where the lesion is smaller, where paralysis or anæsthesia is only partial, and where nursing is efficient.

Myelitis of the lumbar region.—The symptoms in this case are similar to those of an acute disseminated myelitis, limited to that part of the cord which supplies the lower extremities. Theoretically one may suppose that a lesion might be limited either to the uppermost part of the enlargement, in which case certain muscles having connections only with a lower part would be spared; or to this lower segment, in which case the same muscles only would be affected with paralysis. But practically one rarely meets with a case which illustrates this difference. This is probably explained to a great extent by the very limited vertical extent of the portion of the lumbar swelling from which the muscles of the lower extremity receive their nervous supply; that is to say, the upper half of the swelling, measuring probably not more than two inches. In this portion, it is true, the centres for the higher and proximal parts are situated higher than those for the distal muscles; but a myelitis which is not limited (like anterior poliomyelitis) to the gray cornua, but affects gray matter and white indiscriminately, will generally involve a very large number of the muscles of the lower extremity.

Taking a case, then, in which no such distinction can be recognised, the symptoms are again paralysis and anæsthesia of the lower extremities, the anæsthesia reaching no higher than the pelvis, and the paralysis not involving the abdominal muscles. As the spinal centres of the muscles are here concerned, the muscles are flaccid from the first and the reflexes are lost throughout; the muscles soon waste; and the electrical reactions associated with muscular atrophy are obtained—that is, the reaction of degeneration, loss of faradic contractility, and cathodal closure contractions in excess of anodal closure contractions (A.C.C. > K.C.C.) The centres for the bladder and rectum are contained in the lumbar portion of the cord, and accordingly we find paralysis of the sphincters and incontinence of urine and fæces.

Myelitis of the cervical region.—The vertical extent of this portion of the cord is greater than that of the lumbar swelling, and it is more easy, by symptoms peculiar to particular nerves or segments, to recognise differences in the localisation of lesions. A more or less transverse lesion will, of course, cause paralysis of the legs and trunk as well as of the arms below the upper level of the lesion. The affection of the legs will be of precisely the same kind as occurs in dorsal myelitis; that is to say, paralysis, anæsthesia, increased reflexes, with persistence of nutrition and electrical reactions, and in due time muscular rigidity. But in the arms the probability is that the lesion will be of sufficient vertical extent to involve some motor cornua completely, in which case the arms, in addition to paralysis and anæsthesia, will present some muscular atrophy, loss of reflexes, and loss of electrical reactions.

In some cases the arms and legs may be equally involved, so far as paralysis is concerned; in others the arms are much paralysed, and the legs but little.

There are, however, symptoms peculiar to a cervical lesion, due to the implication of special centres or nuclei. The most important of these is paralysis of the diaphragm, which is supplied by the phrenic nerve.

If the lesion be as high as this the result is generally disastrous, for though it is true that patients may live in spite of paralysis of the diaphragm, and even recover from it, especially in cases of peripheral neuritis, the symptom in the case under consideration is generally added to, or associated with, paralysis or paresis of the intercostal muscles, the nervous supply of which arises from the cord lower down, and the upward communications of which are contained in the pyramidal tracts passing through the diseased area. The result on the lungs is stagnating circulation at their bases, œdema and accumulation of fluid in the smaller tubes, dyspnœa, and cough which is unequal to clearing the bronchial tubes, because the respiratory muscles cannot draw in enough air, nor the abdominal muscles expel what there is, with sufficient force. This difficulty soon becomes worse when the lesion is seated at or above the centre for the diaphragm, and brings the case to a comparatively early close.

It is well known that the superior cervical ganglion receives cerebro-spinal filaments from the dorsal nerves, and hence from the lowest part of the cervical cord, and from the first dorsal segment. The sympathetic supplies fibres to the ciliary muscle, to the iris, and to some unstriped muscular fibres in the orbit which keep the eyelids retracted. If this region of the cord (*cilio-spinal centre*) be damaged the sympathetic may be paralysed; and this will result in contraction of the pupil and slight closure of the lids, or diminution of the palpebral fissure. Dilatation of vessels from paralysis of the vaso-motor system may occur; and the following symptoms have also been noticed:—dysphagia; hiccough; variations in the pulse, which is sometimes slow, sometimes rapid and irregular; hyperpyrexia; priapism.

Unilateral myelitis.—In many cases, both of disseminate and of transverse myelitis, the legs or limbs of the two sides are unequally affected; so that the paralysis on one side is greater than it is on the other, and very small foci of myelitis may occur in such a way as to cause some isolated symptom such as paralysis of one or two muscles or some localised anæsthesia; but the occurrence of a primary acute inflammatory lesion, occupying half the thickness of the cord, involving both gray and white matter, and yet strictly limited in extent by the middle line, must be exceedingly rare. As a transverse lesion it appears to be more likely than as a diffused vertical lesion; and still more so in the form of two or more lesions above one another, together cutting all the vertical nerve-paths. These remarks do not apply to anterior poliomyelitis, which is often absolutely unilateral, but to the group of diffuse myelitis in which white and gray matter are involved together.

The symptoms of a unilateral lesion of the spinal cord have been largely ascertained from experimental sections; from some cases of traumatism; and from cases of tumour pressing upon or growing in one side of the cord.

The peculiarities are dependent largely upon the fact, contested, it is true, but still based upon a good deal of evidence, that the sensory fibres for the most part decussate immediately after their entry into the cord, and pass up the opposite side of the middle line to the encephalon.

The following propositions are laid down by Erb and Brown-Séquard. Supposing the lesion to be on the dorsal region, there are—(i.) In the parts supplied by nerves from the segment concerned, (*a*) on the side of the lesion, paralysis, diminished reflexes, atrophy of muscle, loss of electrical reactions or reaction of degeneration, loss of muscular sense, anæsthesia in the form of a transverse band, and above the latter a band of hyperæsthesia; (*b*) on the side opposite the lesion a band of anæsthesia, and above it a band of hyperæsthesia. (ii.) In the parts below the lesion of the cord there are—(*c*) on the side of the lesion, paralysis, retention or increase of reflexes, no muscular atrophy, no loss of electrical reactions, diminished muscular sense, no cutaneous anæsthesia, but, on the contrary, hyperæsthesia, and vaso-motor paralysis; (*d*) on the side opposite to the lesion there is anæsthesia continuous with the band at the level of the lesion, while the motor relations of the limb remain good, or are at most but little affected.

Some differences will appear if the lesion be in the cervical or lumbar region; the upper limit of the anæsthesia and the band of hyperæsthesia, if present, may thus show relations to the known distribution of sensory areas over the upper or lower limbs respectively.

Central myelitis.—This is often described as a very serious form of acute myelitis, on account of the rapid manifestation of the symptoms, the rapid atrophy of the muscles, and the acute and extensive bedsores. But it is clear that the name has been given by various authors to various things. For instance, Dujardin-Beaumetz, in his treatise on acute myelitis, refers this disease to three principal types, of which the third is acute myelitis of the anterior cornua, and may therefore be set aside. The others are "central or generalised myelitis" and "localised or focal myelitis." He then proceeds to describe "acute central generalised myelitis," attributing to it all the rapid onset, acute course, and destruction above indicated. Hallopeau (17, 18), writing, it is true, of chronic diffuse (or interstitial) myelitis, and of sclerosis, uses the word "peri-ependymal" (péri-épendymaire) as synonymous with "central." Many of the cases he reports are unaccompanied by notes of autopsy; and many that he collects as examples are cases either of syringomyelia or of central glioma. He recognises that the importance of the lesion is proportionate to the extent of its invasion of the gray matter, and not of its proximity to the centre of the cord; and, indeed, at the end of his earlier article, he suggests that so long as the lesions remain limited to the immediate neighbourhood of the central canal, they are unrevealed by symptoms.

Lastly, the word "central" is sometimes used to mean the implication of the gray matter, presumably of the posterior and commissural gray matter, as well as of the anterior gray matter; now as the affection of the latter alone constitutes anterior poliomyelitis, we put it aside for separate consideration elsewhere (p. 186).

It can be readily understood that an inflammation of considerable vertical extent involving the gray matter will bring about the acute and disastrous condition with which this disease is credited; but it must be allowed that the term "central" is not a suitable one to apply to the gray matter in its full transverse extent. General or diffuse poliomyelitis would better describe the condition. Further, as regards the symptoms, if an acute destruction of the gray matter take place, there can be little difference in the results, whether the white matter be involved at the same time or not; that is to say, it would be impossible to distinguish the variety before us from extensive disseminated myelitis.

Blocq says that the same dissociation of the sensory phenomena may occur, as is seen in cases of syringomyelia, usually a chronic disease; and more than one author refers to a case by Vallin, in which an acute myelitis of the gray matter was accompanied by suppurative arthritis of the knee-joints. But, as the patient obviously died of pyæmia, with sloughing bedsores, purulent meningitis, and abscesses in the lungs, the dependence of the lesions in the knee upon the poliomyelitis seems more than doubtful. On the other hand, the arthropathics of locomotor ataxy and syringomyelia are well known. Gull has described recovering paraplegia associated with indications of synovitis; and hence a connection may be considered established between these conditions.

Peripheral or Cortical myelitis.—This is undoubtedly in the majority of cases a condition which accompanies meningitis and is secondary to it; and probably in acute cases the symptoms referable to the spinal lesion would not be distinguishable from those which were due to the meningitis. Whether they are mainly due to one or the other element in the meningomyelitis, the symptoms which may arise consist of pains in the spine and limbs, cutaneous and muscular hyperæsthesia, weakness or pronounced paralysis of the limbs corresponding to the portion of the cord involved, contraction of muscles, and exaggerations of reflexes. The muscles are said not to atrophy, and the sphincters to be spared.

Duration and terminations.—Recovery from pronounced myelitis is by no means common, and is likely to occur only in cases of very moderate severity. The great majority of cases are either fatal in the course of weeks or months, or lapse into a condition of incurable paralysis, and are then called cases of "chronic myelitis"; a name which is then justified only by the probability that sclerotic changes in the acutely damaged part may be going on for a long time after the onset.

A case of acute disseminated myelitis may be fatal in from five or six days to the same number of weeks. Less extensive and more localised myelitis may last from twelve months to two years; and in others, finally, life is spared although the lesion persists.

The dangers of myelitis are the implication of the cervical region of the cord and the respiratory muscles, and the occurrence of cystitis and of bedsores. Hence any myelitis is dangerous in proportion to the extent to which it involves the cervical cord on the one hand and the lumbar cord on the other. An extensive disseminated myelitis is quickly fatal, because it involves the cervical region, and causes paralysis of the diaphragm, with consequent œdema of the lungs and broncho-pneumonia. The intense cystitis and the bedsores, which may rapidly form if the utmost care be not used, are probably but slightly contributory in these cases.

Similarly, among circumscribed cases the cervical and the lumbar lesions are most likely to be fatal. As far as the cervical region is concerned, the condition is practically beyond control if the nuclei of the phrenic nerves are invaded; but in the case of the lumbar cord, the fatal result is mostly the result of septic infection from the bladder or the bedsore, and with proper care the occurrence of infection may be postponed indefinitely; or, through relaxation of precautions, may take effect at any stage in the course of the illness.

Diagnosis.—This has, first of all, to be considered with regard to the discrimination of myelitis from other diseases, whether affecting the spinal cord or other parts. Acute myelitis is most likely to be confounded with the following:—spinal meningitis, hæmorrhage into the spinal cord, meningeal spinal hæmorrhage, multiple neuritis, disseminate sclerosis, and hysteria.

Acute spinal meningitis occurs, as an apparently spontaneous disorder, much less frequently than does myelitis. It forms part of epidemic cerebro-spinal meningitis, or of tuberculous meningitis, or arises from the invasion of inflammatory lesions from the side of the bony canal. When associated with cerebral inflammation its symptoms may be masked; but occurring independently it is characterised by symptoms of irritation of the nerve-roots, both sensory and motor. Hence there are pains, both local and radiating, hyperæsthesia, and muscular spasms and rigidity. In a later stage there will be paralysis, but the earlier signs may be distinctive. Febrile reaction is likely to be more pronounced than it is in at least a localised myelitis.

Hæmorrhage into the spinal cord is frequently difficult to distinguish from the more acute cases of myelitis. What is specially characteristic of it is a sudden onset with severe localised pain; but these may be different only in degree from what sometimes happens in myelitis, and the later manifestations are almost identical; namely, paralysis and anæsthesia with the conditions distinctive of the different localities already described. Recovery or improvement may ensue, or permanent lesion with secondary degenerations in both cases (*vide* p. 48).

A further difficulty is suggested by the fact that a hæmorrhage will set up surrounding myelitis, and that hæmorrhage may take place into an already inflamed focus.

Spinal meningeal hæmorrhage.—The diagnosis depends here upon the relatively sudden onset, together with the presence of the symptoms which are regarded as characteristic of surface lesions; namely, those pointing to implication of the nerve-roots. The primary effects are those of irritation, such as we see in meningitis; pain at the seat of the lesion; pain and hyperæsthesia in the course of the nerves arising therefrom; and muscular spasm, also both local, causing rigidity of the back, and more widespread, as in the muscles supplied by nerves arising from the part. After a time, the muscles are paralysed, but the early signs will have been sufficient to distinguish the condition from myelitis.

Multiple peripheral neuritis.—In its definite forms this affects all four limbs, and would therefore be confounded rather with a disseminated myelitis than with a localised lesion. A pronounced paraplegia involving the legs and leaving the arms free can very rarely be a multiple neuritis. Commonly also multiple neuritis, as, for instance, when it is the result of alcohol, of septicæmia, or other poison, is slower in its progress, and to that extent different from acute myelitis; yet multiple neuritis sometimes advances rapidly, as in a case published by myself, in which weakness of the arms and legs was noticed four days after numbness in the fingers and feet; five days later the paralysis was considerable. Moreover, cases of Landry's paralysis, some of which are in all probability multiple neuritis, show a rapid extension of paralysis (*vide* vol. vi. p. 694).

But returning to the more definite cases, we see in multiple neuritis an almost simultaneous occurrence of numbness and tingling in all four extremities, sometimes with pain and hyperæsthesia, weakness of the muscles going on to paralysis in all four limbs, but beginning often or more pronounced in the extensor muscles of the legs or feet; the loss of all reflexes, superficial and deep; comparatively rapid occurrence of wasting in the muscles, with loss or modification of the electrical reactions in the direction of degenerative reaction. What is distinctive of neuritis is that the symptoms begin in all four extremities at the same time, the numbness affecting the hands and feet together; whereas in myelitis the anæsthesia, beginning in the feet, spreads to the legs, thighs, and trunk, and does not affect the arms till the whole of the trunk is involved. Similarly in neuritis the muscles of the forearms and legs may be paralysed together, while the more proximal muscles are little or less affected. If in addition, as sometimes happens, the facial muscles are affected, and the skin of the face is anæsthetic, the diagnosis of neuritis is very positively confirmed. Another important distinction is that in neuritis the bladder and rectum are as a rule spared; whereas in a case of extensive myelitis, which on other grounds might be confounded with neuritis, paralysis of the bladder and rectum is practically constant. Tenderness of the muscles, especially the calves, and of the exposed nerve-trunks such as the external popliteal and ulnar nerves, is another common sign in neuritis not found in myelitis.

Landry's paralysis.—The nature of the cases described under this name is even yet uncertain (*vide* vol. vi. p. 694). Some writers, for instance, Blocq and Grasset and Rauzier, include them under myelitis, as an acute, diffuse, or acutely spreading (envahissante) form; but I think the reasons advanced by the late Dr. Ross (25) for our regarding many of the cases at least as multiple neuritis are very strong indeed. In neither case should a separate paragraph on the differences between it and myelitis be required: in the one case it is not different; in the other the distinctions between myelitis and multiple neuritis, already described, should cover the ground. But so long as the disease is allowed a separate place something must be said here of its diagnosis from myelitis. The synonym of Landry's paralysis—acute ascending paralysis—is of course not distinctive, and does not indicate all the special features of the disease: a disseminated myelitis is often an acute ascending paralysis, and it is from these disseminated or diffuse forms of myelitis that Landry's paralysis has to be distinguished, by the absence or comparative insignificance of the sensory phenomena, and by the freedom of the bladder and rectum. The distinctions seem small, but it will be readily seen that a really disseminated myelitis must, and in fact does, involve the sensory tracts largely, and the functions of the bladder and rectum seriously; and that if these escape the lesion must be one involving the motor functions alone (excluding the viscera), and must therefore, as a myelitis, be limited to the anterior cornua alone. Now an inflammation limited to the anterior cornua, and rapidly ascending from lumbar to cervical region, has not been found to explain these cases, and indeed must itself be a very rare event.

From a circumscribed transverse myelitis Landry's cases are easily separated by the rapid spread of the symptoms in an upward direction till the chest is involved, as well as by the features, above mentioned, in which it differs from disseminated myelitis.

Disseminate sclerosis. — Another disease with which symptoms of myelitis may be associated is disseminate sclerosis; that is to say, disseminate sclerosis may begin with acute paraplegic symptoms; but in course of time the intention tremors, staccato speech, and nystagmus will probably show the nature of the case.

Hysteria.—Hysterical paralysis and functional weakness of the lower extremities are of frequent occurrence, and often difficult to distinguish from the milder forms of myelitis. The female sex, youth, and a previous history of hysterical outbreaks may serve to caution us against too hasty a diagnosis of myelitis. More often, I am afraid, the caution is wanted on the other side; and it must not be forgotten that young women with hysterical tendencies are by no means prevented thereby from having organic lesions of a serious kind.

Hysterical paralysis is sometimes distinguished by the mode of its onset; by its arising immediately in connection with some emotion, or mental excitement. Often one leg is affected alone, and the patient tries to walk, but holds the weak leg constantly behind the other, dragging it up to the other, but never bringing it in front: the muscles of the affected side, instead of being relaxed and helpless, are generally held rigid during the process. But this monoplegic form is little likely to be confounded with the usually bilateral paralysis of genuine myelitis. In case of a bilateral affection the important points to note are that in purely hysterical conditions there is no spastic rigidity of the kind previously described, brought on, that is, by various external stimuli, as by handling and passive movement (*vide* art. "Hysteria" in following volume). Muscular contractions of a more voluntary kind can often be elicited, in either of the limbs supposed to be paralysed, by lifting the leg some distance from the bed while the patient's attention is distracted by various questions.

The limb will often then be supported, or fall slowly to the bed in a series of short drops, alternating with short rests for which the elevating muscles are obviously brought into contraction. Again, any pronounced alteration of the reflexes is opposed to hysteria. A slight ankle clonus of short duration, and unsustained, sometimes occurs in hysterical people; but a continuous strong clonus of the ankles, with marked knee clonus, is against a purely functional origin for the weakness. Similarly entire abolition of the knee-jerk in one who had it normally, and the exceptions, if any, are exceedingly rare, means organic disease. In hysteria, also, there is rarely specific wasting of the muscles or loss of electrical reactions; bedsores do not occur, nor is there incontinence of urine or fæces. Retention of urine, on the other hand, is often present.

Diagnosis of the site of myelitis.—The determination of this point must, of course, depend upon a knowledge of the relations which the functions of motion and sensation in different parts of the body bear to the different segments of the spinal cord.

A great deal of work has been recently done in this subject, and as a result of experiments and observation of accidents and diseases it has been ascertained, with a certain amount of agreement among different observers, what sensory areas on the surface of the body, and what muscles or groups of muscles, correspond to the roots of the spinal nerves.

The relations of the spinal cord segments to sensory areas, muscle groups, and reflex phenomena, are given in a previous article (vol. vi. p. 865).

In the majority of cases of myelitis the anæsthesia offers the best means of determining the upper limit of the lesion, because it is often very definite, and sharply outlined at its upper margin, which forms a line running transversely round the body, and much more truly horizontal than our knowledge of the anatomical course of the nerve-trunks would lead us to expect; whereas from the anatomical arrangement of the muscles in long vertical bundles and continuous planes, some of which, especially in the abdomen, receive fibres from several nerve-roots, the separation of the parts affected from those spared is much more difficult.

The determination of the lower limit of an area of myelitis is less simple. Voluntary motion and sensation below the lesion are cut off entirely by any transverse lesion, of whatever vertical extent; but the integrity of parts below the lesion is recognised by the persistence of reflexes, of the nutrition of the muscles, and of the electrical reactions. These reflexes include those of the bladder and rectum. By such means one can recognise—for example, in a dorsal lesion—that the lumbar swelling is virtually intact; but any more accurate estimate is rarely possible, because the skin reflexes are few in number and operate over large areas, and the discrimination of muscles supplied by the intercostal nerves is difficult.

Prognosis.—The duration and terminations of myelitis have already been given, and the prognosis or the probable outcome of the disease in any case has been there indicated.

Some cases undoubtedly end in recovery, but how are we to distinguish these in good time from those which do not? It must be allowed that there are very few means of forming a safe forecast. The more acute and the more extensive the lesion the less likely is recovery to take place. A severe disseminated myelitis is very likely to be fatal within a few weeks or months. Yet if its ascending progress stops, or is arrested, if the bladder can be kept healthy, and if bedsores can be avoided, then death may be staved off indefinitely. However, again, the more disseminated the lesion, and the more the lumbar centres involved, the less likely is it that the necessary extreme amount of care and watchfulness will be exercised; and death mostly occurs within two or three years. In cases of myelitis, restricted to the dorsal region, the integrity of the lumbar centres is in favour of the patient; and life may be prolonged for many years, even if no actual recovery take place.

There are few features, if any, from which one can say that recovery will certainly ensue. It is probable that in no case can such a favourable course be predicted. It is only when the paralysis is actually beginning to subside that one can look forward to recovery, and even then the progress may be checked for some quite unknown reason. Amongst cases recorded as arising in the early stages of syphilitic infection (secondary syphilis), and treated by antisyphilitic drugs, recovery has not infrequently been witnessed. And I should be inclined to look hopefully upon other cases which could be definitely regarded as secondary results of some infectious process.

The paralysis associated with spinal caries (compression myelitis) is well known to present a more favourable prognosis than many other forms. Even after months of incapacity, with paralysis and increased reflexes, the consolidation and cure of the spinal lesion is very likely to be accompanied by complete recovery of power in the affected limbs (see vol. vi. p. 869).

Treatment.—The treatment of acute myelitis has gone through all the phases known in other acute visceral inflammations. Local depletion, heat, cold, vesication, counter-irritation, and mercury are or have been employed with results apparently less successful than can be shown for acute pneumonia or acute nephritis under similar methods—an appearance which is probably explained by the greater vulnerability of nerve tissue as compared with that of the lung. We must at any rate allow that as yet there is no known specific for myelitis as such; and it can only be matter of hope in the future that, as the infective nature of different cases of myelitis is demonstrated, the special antidote of each infection may come to be known, and be found efficacious in improving or curing the condition of the cord. This is in part realised as regards syphilis, and preventively as regards hydrophobia, in so far as a paralysis is the last stage of this disease; but beyond this little or nothing can be said to have been accomplished.

The old "antiphlogistic" methods already referred to aim chiefly at modifying the state of the circulation in the inflamed organ. But it is doubtful, on the one hand, if the changes in the vascularity of the organ are the primary lesion; and on the other, if they are, whether the methods adopted modify it to any material extent.

The pneumonic lung is apparently much more accessible than the spinal cord to applications of various kinds upon the skin; and yet it is difficult to measure how far heat, cold, blisters, and cupping have any direct influence upon the course of an ordinary acute lobar or croupous pneumonia.

However, the discussion of this subject belongs rather to general therapeutics. If in the desire to omit no method that could by any possibility be useful, I had to make a choice of one of them, I should myself not go farther than the use of cupping, in the earliest stage of the complaint, to such part of the spine as might be acutely painful; and in other cases would apply cold, in the form of ice bags, or heat, in the form of hot fomentations or of bags containing hot sand. I believe there is more to be said for cold than for heat in the local control of inflammations; on the other hand, the suggestion that in cases believed to arise from cold, hot applications should be used in the first stage, seems at present reasonable. Hot applications are generally more agreeable to the patient. When using very hot applications, the stronger counter-irritants or blisters, the possibility of inflicting considerable injury to the skin must be borne in mind.

The position of the patient is of some importance. Rest is, of course, essential, and the most complete rest is undoubtedly given when the patient lies flat on his back; but in this position the spinal cord is nearly in the most dependent part of the body, so that there must be a tendency to gravitation of the blood into its neighbourhood, and, to that extent, an increase of the high vascularity which attends acute inflammation. This may be neutralised, or diminished, by placing the patient on the right and left side alternately; or even in the prone position for a longer or shorter time. Either of these positions, if it can be borne by the patient, facilitates the requisite applications to the spine. As soon as possible a water-bed should be procured, in order to lessen the risk of bedsores; and the greatest care and attention to cleanliness are required throughout the case (*vide* vol. i. p. 430).

The *diet* must be arranged on the same principles as in any other acute or febrile disease (vol. i. p. 385). With an acute onset, whether fever be well marked or not, it is right that the patient should be restricted to liquid diet; namely, milk, beef tea, mutton broth, meat essences, and similar foods. And if the fever persist, so long must milk or slop diet be continued. When the fever subsides, a farinaceous diet may be supplied, such as bread and butter, bread and milk, custard and milk puddings; and when the illness has lost its acute features solid foods may be given, so far as they are perfectly plain, and in quantity adapted to the needs of a perfectly inactive individual.

The treatment by *drugs* can scarcely be called satisfactory. It is right to see that the bowels, the kidneys, and the skin are acting normally;

and free excretion from these organs may help to lessen the vascularity in the diseased part. The use of other drugs is largely based on speculative grounds. Ergot has been given for its contractile effects upon the spinal vessels; if used, it must be freely administered in doses of one or two drachms of the liquid extract or infusion. Mercury, in the form of perchloride or of calomel, has been much used both in antiphlogistic times and more recently; and, for its supposed powers of absorbing inflammatory products, iodide of potassium has been employed; though rather in later than in earlier stages.

The value of these remedies is at best uncertain except in some of those cases which can be safely attributed to syphilis. On the hypothesis of the infective origin of the disease, such antiseptic agents as quinine, salol, salicylic acid, and β-naphthol have been given; but seeing how little they control other infectious diseases, even those which have their primary seat in the intestinal mucous membrane, we cannot expect much from them in the present malady.

Undoubtedly the most important considerations in myelitis, from its earliest stage, are the care of the bladder and the prevention of bedsores. If retention of urine occurs the bladder must be emptied promptly by means of a thoroughly aseptic catheter, and the process must be repeated at least twice daily as long as there is any tendency to accumulation of urine. In the case of a circumscribed transverse myelitis, when the bladder is often emptied spontaneously and involuntarily, whether unconsciously or not, the risk of cystitis is less. If from any cause, either before or after the case comes under treatment, micro-organisms have found an entrance, and cystitis has begun, the urine must be withdrawn, the bladder should be washed out twice daily, and some antiseptic fluid, such as Thompson's fluid, introduced after each washing. Boric acid (10 or 15 grains), salicylic acid (15 grains), or salol may at the same time be given internally to diminish the alkalinity of the urine.

The *prevention of bedsores* requires the utmost cleanliness on the part of the nurse. It must be remembered that, in a case where anæsthesia is present as well as paralysis, many things favour the occurrence of lesions of the skin of the dependent part. The vitality of the skin is lessened by loss of nerve influence, by the continued pressure of the weight of the body upon it, and by the diminished circulation which that pressure entails; the patient is unable to feel any irritating matters in contact with the skin, or to realise that his skin is wet with urine, or soiled with fæces; again, if he knows these facts, he cannot shift himself in the bed so as to obviate or remove them. From the presence of the worst possible irritants he is in constant danger; that is, from the dribbling of urine from the over-distended bladder, or the unconscious passage both of urine and of fæces. All these evils must be prevented; they must be met by the use of a water-bed, the occasional turning of the patient to one or other side, so as to relieve the pressure on any one region, the frequent examination of the bed and the patient, and the anticipation of any call of the patient for help, when he feels, as in some stages he may, the desire to be relieved.

In addition the lower parts of the back should be kept scrupulously clean, and after washing should be dabbed with spirit lotion or eau de Cologne, and dusted over with oxide of zinc or calamine powder. If a slough have formed it may be dressed with solution of boric acid, or dusted with iodoform powder; and to the sore which remains after its separation one of several antiseptic preparations should be applied:—lotions of boric acid or corrosive sublimate (1 in 500), a mixture of equal parts of unguentum resinæ and balsam of Peru, iodoform, boric ointment, or salicylic ointment (*vide* vol. i. p. 431).

In later stages of myelitis, tonics, such as quinine, the mineral acids, nux vomica, bitter infusions, and strychnine, may be given.

Electricity is a remedy not as a rule applicable in the early stage of the disease. Whether, and to what extent, the seat of the lesion in the spinal cord is actually irritated by the passage of a current in the limbs, may be open to question; but at any rate the stimulation of the muscle to contraction can be of little service in the early stages, or until it is known that the connection with the motor centres is likely to be restored. Its use in the later stages depends upon the course the disease takes; that is, whether it is lapsing without improvement into a chronic condition, or is gradually improving, however slowly.

The former case will come under the head of chronic myelitis. In the latter case the limbs will probably not show much rigidity, because much rigidity is generally associated with that pronounced secondary degeneration of the lateral columns which is the indication of a serious and often incurable transverse lesion. If rigidity be present the muscles are in constant or frequent contraction, and their further stimulation by electricity seems unnecessary and undesirable. If on the other hand the muscles are not rigid, or are flaccid, the electrical current may supply the stimulation, which is deficient both in force and frequency as coming from the spinal centres. But it must be understood that this does little more than maintain the nutrition of the muscles by causing their physiological contraction, and has little (if any) influence upon the centre of the disease in the spinal cord. Electricity is not indeed directly curative, but is only an adjuvant to other treatment, keeping up the nutrition of the limbs to as high a pitch as possible.

The faradic current is the form more generally used; but where the contraction of the muscles is desired, and can be effected, whichsoever of the two currents that will cause this contraction may be employed.

Under similar circumstances massage may have a similar usefulness, and may be practised with advantage (vol. i. p. 373).

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CHRONIC MYELITIS

This name is generally taken to signify cases of myelitis of long duration; both those which, beginning as acute cases, have never improved, and those of slow onset from the first. Probably the former group is much more numerous than the latter. On the other hand, it should be restricted, as was acute myelitis, to the group of cases in which the inflammation is primarily interstitial and diffuse, and not selective either of parenchymatous structure on the one hand or of particular anatomical tracts of nerve fibres on the other. Thus the system-diseases—locomotor ataxy, Friedreich's disease, and so forth, whether they be mainly inflammatory, or, as is more probable, primarily degenerative, as also disseminate sclerosis and syringomyelia, will be dealt with elsewhere; and those forms only will be described which in distribution are of the kinds described under acute myelitis, but of which either the onset is slow, or the course protracted over several months or years.

Etiology.—This need only be referred to in so far as it concerns the cases of insidious or slow onset. Difficult as it often is in cases of acute myelitis to attribute the disease to a definite cause, it is still more so in the case of a chronic lesion; and although many of the same factors are believed to be in operation, it is clear, nevertheless, that much light is still needed in this part of the subject.

The factors which are believed to operate are the following:—

Heredity in the form of a neuropathic tendency; cold, operating repeatedly rather than on one single occasion as in acute myelitis; fatigue; venereal excess; emotional disturbances. It is needless to remark on the missing link between these etiological factors and the result, a missing link which will probably be found some day in the form of a micro-organism or derivative toxin. Indeed the other causes which have still to be enumerated are brought with no great difficulty under these or allied classes; they are, first, local inflammatory conditions, such as diseases of the bone, chronic meningitis, tumours of the spine; secondly, the infectious diseases, especially syphilis and typhoid fever; thirdly, chemical poisons, especially lead, arsenic, phosphorus, mercury, and alcohol.

Pathological anatomy.—The distribution of the lesions may be considered under the same heads as those of acute origin. Thus there is a *transverse* or *focal* myelitis, and this is by far the most frequent form of chronic myelitis. A *disseminated myelitis* which becomes chronic, or is so from the first, is a "disseminated sclerosis"; and it is perhaps doubtful if any condition of disseminated myelitis, which is more extensive or more diffuse than what is characteristic of "sclérose en plâques," can occur without either killing the patient or giving rise to the symptoms which distinguish this disease. It is well known that "sclérose en plâques" may begin acutely. On the other hand, von Leyden and Goldscheider recognise sclerosis as the only possible result of chronic myelitis, and describe chronic transverse myelitis only as a spinal form of multiple sclerosis. Oppenheim and Bruns and Windscheid hold a similar opinion.

Rarely is seen an annular or peripheral myelitis, in which the surface of the cord is so affected as, on transverse section, to present a ring of sclerosis. Mostly this localisation is associated with inflammation of the meninges, or perhaps determined by it, and the disease is really a meningo-myelitis.

The other possible division is the *central* myelitis; but it has been shown above that many of the chronic lesions of this form, described under the heading of chronic peri-ependymal myelitis (Hallopeau), would now be classed as syringomyelia, and are accompanied by the symptoms of that disorder (*q.v.* p. 22).

To the naked eye the affected portions of the cord present a pinkish gray appearance, so that when they occur in the white columns they usually resemble the healthy gray substance. In consistence they are often harder than normal, and, if of any extent, may bring about some shrinking or diminution in the thickness of the cord. Occasionally they are softer; and, in cases which have begun as acute myelitis, the softer acute stage may persist for some months before it is replaced by induration.

Under the microscope, the distinguishing feature of the more chronic indurated portion is the excess of neuroglial connective tissue, consisting either of a distinctly fibrillated substance, or of a more amorphous homogeneous substance containing nuclei in more or less abundance. The neuroglial cells with radiating processes may also be in great number. In the white matter the nerve-fibres have generally undergone considerable atrophy, though in earlier stages some may still be seen swollen; and in the gray matter, which equally shows the fibrous development of this connective tissue, the nerve-cells become pigmented, lose their processes, alter in shape and size so as to be small or globular or angular, and finally may disappear.

In most cases the walls of the vessels are much thickened, and the connective tissue adjacent to them may be more hypertrophied than elsewhere. In meningo-myelitis the connective tissue bands seem to take origin in the meninges, and pass thence into the substance of the cord.

In the softer and less indurated lesions the connective tissue development is proportionally less, and the granule-corpuscles, which are so numerous in acute lesions, may be present.

Secondary degenerations occur in chronic myelitis in accordance with the position of the lesions, and in proportion to their transverse extent.

Symptoms.—A complete account of the symptoms of chronic myelitis would repeat much that has been said of the acute forms. Where the chronic stage has succeeded the acute the symptoms persist with but little change. In the majority of instances the lesion is in the dorsal cord; and more or less complete paralysis, with some degree of anæsthesia, increased reflexes, and spastic rigidity of the limbs, are the prevailing features. If the lesion be in the lumbar region the limbs may be flaccid, and the bladder may be seriously implicated. But there are two conditions which, however they may be brought about, militate against a chronic course; these are profound anæsthesia—which in neglected cases may lead to extensive bedsores, and any condition of the bladder which may promote cystitis; for by either of these incidents life is likely to be shortened before any long time can elapse. In cases of primarily chronic myelitis the onset may be slow and insidious; months or years being required for the full establishment of the case. Sensory symptoms are often the first to be noticed — tingling, formication, anæsthesia, and analgesia; then weakness of the legs is observed, perhaps one leg being affected before the other. Later, in the case of a dorsal lesion, the legs become rigid and spastic, the knee-jerks are increased, and ankle clonus appears. Of course there may be the greatest variety in the extent and severity of the symptoms; the paralysis may be slight or complete, the anæsthesia is very often comparatively slight while the loss of motion is greater, the spastic rigidity may be only just detected on manipulation of the limbs, or on the other hand it may be almost constantly present, relaxing only at times, and brought on or aggravated by the slightest movement, or a touch, or perhaps even by the thought of movement on the part of the patient. In this extreme form it is common for the legs to be bent so strongly at the knees and hips that the thighs are near the abdomen, and the heels near the buttocks; or with less extreme flexion the two thighs partially cross each other by the force of the adductors. The patient is quite unable to straighten the limbs; and if during sleep, or by gentle traction, the limbs should become extended, they are quickly flexed again on the receipt of fresh stimuli on the surface.

If the lumbar enlargement is affected some atrophy of muscles is likely to occur, and the reflexes may be lost: if the cervical enlargement, the arms will be paralysed, with partial or more complete wasting of muscles. In either case the lesion may be unevenly distributed, involving white columns as well as gray substance; and spastic rigidity may be associated with the muscular atrophy.

The special functions of the cervical and lumbar regions, in contrast with those of the dorsal region, may be disturbed in chronic myelitis as they may in acute: thus in the latter case the rectum and bladder will suffer; and in the former may occur a number of symptoms concerning the eye, and the functions of respiration, circulation, and deglutition.

In the lower part of the cervical spine is situated the cilio-spinal centre: irritation of this results in dilatation of the pupil and pallor of the face; paralysis or destruction in the converse conditions. Other symptoms which have been noted are cough, dyspnœa, hiccough, slow pulse, gastric crises and dysphagia.

Diagnosis.—Most cases, and especially those which have begun abruptly, may be tried by the tests given in the section on acute myelitis.

Primarily chronic cases must be distinguished from the systemic diseases of the spinal cord. These are often recognised by some positive sign which corresponds to the peculiar limitation of the disease. In disseminate sclerosis the oscillating movements of the limbs on voluntary action, the nystagmus, and the staccato or scanning speech are distinctive; though in late stages the lower extremities may be paralysed, anæsthetic, and rigid, so as closely to resemble a myelitis of the dorsal region. In tabes dorsalis, ataxic paraplegia, and Friedreich's disease, incoördination is a marked feature; in the first there are the pupil-phenomena, the lightning pains, the absent knee-jerk, and the retention of muscular power, at least for a long time; in the third there is generally the hereditary history or family association, and nystagmus and difficulties of speech are present. Syringomyelia and poliomyelitis anterior chronica are characterised by muscular atrophy; and though we have seen that muscular atrophy may be present in chronic myelitis of the cervical or lumbar region, it is in myelitis generally subordinate and secondary to the paralysis; whereas in poliomyelitis chronica atrophy is the first sign of disease, and usually follows a very definite course in its selection of the small muscles of the hands. Syringomyelia, again, is distinguished by the peculiar manner in which sensation is affected, the transmission of ordinary touch sensations being unimpaired, while the patient is insensitive to pain and change of temperature. Moreover in this latter disease the joints are often invaded by chronic and destructive changes.

There remains primary spastic paraplegia, in which muscular weakness, spastic rigidity, and increased reflexes are attributable to a primary degeneration or sclerosis of the pyramidal tracts in the lateral columns. So far conclusive pathological proof of this connection has not been provided; but such cases, clinically, can be recognised and are distinguished from the *secondary* spastic paraplegia of myelitis by the persistence of the sensory functions, and of the functions of the bladder and the rectum. A more common mistake is to regard a spastic condition as primary; and to fail to seek for the evidence—such as anæsthesia, and rectal and vesical troubles—of some pathological condition outside or above the pyramidal tracts.

Amyotrophic lateral sclerosis, in which muscular rigidity and weakness are associated with muscular atrophy, is also distinguished from a diffuse or transverse myelitis by the integrity of sensation, and of the functions of micturition and defæcation.

If it is recognised that the lesion is a chronic transverse myelitis, there is yet something to be learnt, positively or negatively, as to its causation. Especially with regard to treatment, it is desirable to know whether the lesion be due to compression by tumours, as in caries of the spine (compression-myelitis), or to syphilis.

Compression-myelitis will as a rule be accompanied or preceded by some symptoms referable to the primary lesion, such as tumour, or Pott's disease, which has caused it (vol. vi. p. 854). For instance, in Pott's disease we have the familiar prominence of the vertebral spine at the affected spot; and in no case of apparently chronic transverse myelitis should an examination of the spinal column be neglected. In earlier stages local pain may be present, and tenderness to pressure, blow, or vertically delivered shock may be elicited; otherwise the symptoms of the lesions may be identical. But it is common to find in cases of compression from Pott's disease that the motor symptoms are more prominent than the sensory; a fact which is explained by the seat of the disease in the bodies of the vertebræ rather than in the laminæ, so that the anterior part of the spinal cord suffers first.

In reference to syphilis attention must be called to the attempt which has been made to distinguish a form of chronic myelitis due to this disease. Erb has described a **syphilitic spinal paralysis**, of which he regards the following as characteristic signs: (i.) the usual symptoms of spastic paraplegia; (ii.) marked exaggeration of the deep reflexes; (iii.) muscular contractions, slight as compared with the reflexes; (iv.) slight but distinct disturbances of sensation and implication of the bladder.

The onset of these cases is gradual, and they show a tendency to improve.

More than half the cases occurred within three years of infection; and though Erb had not verified any by necropsy, he thought the lesion was partly a syphilitic infiltration and partly syphilitic disease of the vessels, leading to myelitis. As to its site it must be a partial transverse myelitis involving the posterior rather than the anterior parts of the cord; that is, the posterior parts of the lateral columns, the posterior cornua, and the posterior white columns. The existence of this type has been recognised by Charcot, Marie, and Muchin. Sachs is unwilling to admit that this is the most important or most frequent form of syphilis of the spinal cord; but from the point of view of the diagnosis of chronic myelitis, it is sufficient if it be allowed that syphilis does sometimes give rise to cases in which, while a transverse spinal lesion is recognised, special features about the case (which features, however, can only be evidences of localisation) suggest that syphilis is the cause.

Prognosis.—This is essentially unfavourable. If sclerosis become established we know of no methods by which this can be removed: on the other hand, acute exacerbations may subside or yield to treatment.

Life may be spared for a long time; but it will be endangered in proportion to the severity of bladder symptoms, and to the liability of bedsores or lesions of the skin from immobility of the patient or contracture of the limbs.

Treatment.—The lines upon which treatment will be directed in ordinary cases are to preserve the use of the limbs as much as possible by the employment of electricity, massage, and moderate exercise, if the limbs are still capable of some movement. Bedsores and cystitis must be prevented. For the direct treatment of the myelitis little can be done. Iodide of potassium, mercury, and arsenic are the remedies chiefly in use: and in cases presumably syphilitic the two former may be pushed with some hopes of success. Local measures are also employed sometimes, and consist of counter-irritation by blisters, and even by the application of the cautery. Such treatment seems more likely to do good in the case of acute exacerbations, which may be treated in the same way as acute myelitis. In all chronic diseases of the kind it is very important to see that all the muscles which retain any capacity be kept in exercise by natural or artificial means, and the disease thus reduced to its lowest terms.

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THE CAISSON DISEASE

A PERSON who has been exposed to a greatly increased atmospheric pressure is liable, on his removal from it, to suffer in proportion to the degree of the pressure, the duration of the exposure, and the rapidity with which the normal pressure is restored.

These morbid results are—in the order of frequency: pain, often very severe, in one or more of the extremities, and occasionally in the trunk; pain in the epigastrium, which may or may not be associated with nausea and vomiting; paralysis, more or less extensive and complete; headache; vertigo, and coma. In rare cases sudden death occurs almost without preceding symptoms.

The pain, which is of a neuralgic character, may be slight and transient, or extremely severe and persistent. It is usually intermittent or remittent. It may come on gradually, increasing in severity until it becomes absolutely intolerable; or it may begin at once in its full intensity. The knees, legs, and hips are most frequently attacked; but the arms or trunk may be the first to suffer. Sometimes the greatest suffering is in the back, and particularly in the lumbar region.

Epigastric pain is of frequent occurrence. If not quickly relieved it is followed by sickness and vomiting. Vomiting may take place without preceding gastric pain, and then is usually accompanied by giddiness or other evidence of cerebral origin.

Paralysis occurs with increasing frequency and completeness in proportion to the degree of pressure and the duration of the exposure to its influence. The lower portion of the body is more liable to attack, but the upper extremities are not exempt. The paralysis is of sensation as well as motion, but it gives no relief to the pain. The part is insensible to pinching or to the prick of a pin, while at the same time it is the seat of extreme suffering. But there is no necessary relation between the pain and the paralysis, as either may occur separately. The paralysis varies in degree, from a slight and transient paresis with some impairment of sensation to complete and permanent loss of motion and sensation in the affected part. Even the minor degrees usually include the bladder.

Symptoms indicating cerebral disturbance of a transient character are often observed, such as headache, double vision, giddiness, incoherence of speech, and occasionally syncope. The skin is often mottled in patches, some of which are veritable ecchymoses; others are the result of stasis in the distended capillaries, and can be rubbed away by persistent friction.

The duration of an attack varies extremely. It may last a few hours, or it may continue for six or eight days. Paralysis may be recovered from in a few days, or be protracted for weeks or months. Death occurs only in cases that are severe from the first; and, except when due to secondary lesions, it usually takes place within three or four days from the beginning of the attack.

Morbid anatomy.—The most common lesion in fatal cases is congestion of the brain or cord, including the meninges. There is usually effusion into the arachnoid. The scalp and the tissues surrounding the spinal column may be engorged. The brain and cord are sometimes softened in spots. Visceral congestions are generally present. The lungs usually show nothing more than hypostatic congestion, though in some cases they are found engorged or œdematous.

Van Rensselaer analyses the results of twenty-five autopsies as follows:—

"Seven of the cases might be termed *foudroyant*, death occurring in from fifteen minutes to nine hours. The spinal cord was found softened in the one case in which it was examined. In five cases the brain was examined; it was congested in two, normal in three. The kidneys, spleen, liver, and lungs were congested in three cases, normal in two. In two of the cases air was observed in the veins. One had marked Bright's disease. One was a drinker, the habits of the others not being stated. The average age of the men was thirty-eight years. The pressure under which they worked varied from two to four atmospheres.

"Six cases were acute, death occurring within a week. The spinal cord was congested or soft in all; the brain and all the internal organs congested in five. The average age of the men was twenty-eight, nearly ten years younger than the first set. In three of these cases there was serous effusion in the spinal canal, and in four extravasations of blood on the dura of the cord.

"In the subacute cases, in which the men died between the first and second week, the spinal cord was found congested in all the cases, and the brain was congested in the three cases in which it was examined. Effusion of serum in the spinal canal was present twice. Cystitis and pyelitis occurred in two cases. The average age of the men was twenty-five.

"The more chronic cases, those in which death took place between three weeks and three months, exhibited similar lesions to the preceding cases, that is, the cord was either congested or softened, the softening appearing usually in the lower dorsal or upper lumbar region; the abdominal organs were for the most part congested. Pyelitis, cystitis, and bedsores were the usual complications. The average age of these patients was twenty-eight years.

From these in many respects defective autopsies it appears that the macroscopic lesions consist in congestion or softening of the spinal cord (in all the cases in which it was examined); congestion of the brain (in 13 cases out of 16); and congestion of most of the internal organs (in 17 cases out of 22)."

I have been able to find five cases only in which a microscopical examination of the cord was made. In three of these, cited by van Rensselaer, death did not take place for 15 days and 2½ months respectively after the attack. Degeneration had taken place, chiefly in the lower dorsal region; but nothing can be inferred from this as to the nature of the primary lesion, for the appearances were necessarily the result of secondary processes.

In two cases at the Presbyterian Hospital, New York, death occurred within twenty-four hours after the attack. At the autopsy the cords were found, both macroscopically and microscopically, perfectly normal in appearance (24).

Pathology.—The pathology of this disease has been the subject of much discussion, and it is probably not uniform for all cases. It seems to be intimately connected with the congestions already referred to, though the direct effect of pressure upon the nerve elements may have a part in the results. Were the latter a prominent agency, however, we should expect to see the disease manifested while the subject was in the caisson, instead of after leaving it. It would not be possible within the limits of this article to discuss fully the various hypotheses that have been put forth concerning this disease. The most prominent is that of François, who suggested that it is due to the liberation of air from the blood in consequence of the reduced pressure, and that this air in bubbles obstructs the smaller vessels. This hypothesis of gaseous emboli was adopted later by Bert (3), who asserted, however, that the bubbles are not of air, but of nitrogen. He supported his views by experiments upon animals, and was able to demonstrate that such emboli are actually formed when a sufficient pressure is employed; but under a pressure less than five atmospheres no such results could be obtained; and even with this pressure, if three minutes were allowed for restoration to the normal, no bubbles could be discovered in the vessels. These experiments seem, therefore, to demonstrate rather that air or nitrogen emboli are not the cause of the phenomena of the caisson disease (25); since these phenomena may occur, and often do, when the pressure is not greater than two atmospheres, and when six to eight minutes are employed in its reduction. Under these conditions we should expect that any air or gas disengaged from the blood would escape through the lungs as it is liberated. Moreover, we must not forget that the symptoms often do not occur for several minutes—or even hours—after coming from the caisson into the open air; during which time, as Bert's experiments show, the liberated gas should have passed off through the lungs.

My own opinion is that the long-continued pressure produces changes in the distribution of the blood; especially in the closed cavities, such as that of the skull and of the spinal canal. The greater the pressure and the longer its continuance the more decided will be these changes, and the less readily will the vessels resume their natural condition when the pressure is relieved. Hence, if the pressure be suddenly reduced, congestions or blood stases result, particularly in the brain and cord, and perhaps also in the cavities of the long bones, which are so often the seat of exquisite suffering. This view is accepted by van Rensselaer.

Causes.—The one cause, without which the disease cannot occur, is *the sudden transition to a lower atmospheric pressure after a prolonged exposure to a higher one.* The extent of the change of pressure required to produce the disease differs greatly in different persons, and is also a matter of habit. Some men will suffer severely from a pressure that ordinarily causes no inconvenience,[1] and many who are susceptible at first become less so as the work progresses. Perhaps the most frequent exciting cause is too rapid a reduction of the pressure in "locking out," that is, in passing from the caisson to the open air through the lock or ante-chamber in which the pressure should be gradually reduced. Indeed, it is probable that if sufficient time were allowed in the lock the disease would never occur. But, obviously, there is a limit to the time that can be so employed, and the best that can be done is to fix a time proportioned to the pressure. This should not be less than five minutes for each additional atmosphere.

Newcomers to the work are universally found to be more susceptible to attack (15). Knapp found that only 10 per cent of the old hands employed at Wyoming were affected, while 35 per cent of the new hands suffered to a greater or less extent.

Fulness of habit, in my experience, adds greatly to the liability to attack. Of 39 men of heavy build employed in the New York caisson, only 3 escaped illness; while of 53 lank and spare men 25 escaped. Of the 39 stout men 8 were more or less paralysed; of the 53 slender men only 2 were paralysed. The deaths, 3 in number, were all of heavy men.

Severe exertion immediately after leaving the lock adds greatly to the danger. Hence, if the ascent be by stairs, the lock should be at the top of the shaft, so that the climbing will be done while in the compressed air instead of just after emerging from it.

The abuse of alcohol has been observed by many writers to increase the liability to attack.

Entering the caisson fasting appears to have been a proximate cause in several instances (14). In these cases epigastric pain and retching were prominent symptoms.

Treatment.—This will depend upon the severity of the attack and the presence or absence of epigastric pain or of paralysis. If neuralgic pain be the principal or only symptom, the chief reliance must be upon the liberal use of an anodyne. Fortunately the pain, though very severe while it lasts, is generally of short duration. It is, therefore, possible to keep the patient during the whole time under the influence of morphine, thus sparing him all extreme suffering. Large doses may be required, as much as half a grain at first, to be followed by a quarter of a grain every half-hour until relief is obtained. If given subcutaneously somewhat smaller doses will suffice.

Working on the hypothesis of the presence of vascular paresis as the chief element in the pathology of the disease, I was led to use ergot to induce contraction of the arterioles, in the expectation that it would relieve the congestion in the brain and cord. The result was not disappointing, and the practice has been followed by others with considerable success. Knapp found it very useful in a number of cases, and it was employed, frequently with good results, at the Presbyterian Hospital, to which cases were brought from the East River Tunnel during the years 1893-94 (24). A teaspoonful of the fluid extract may be given, and the dose repeated in half or three-quarters of an hour unless the pain is relieved.

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[1] This predisposition suggests an analogy with the readiness with which many persons appreciate a fall of the barometer, feeling it, as they say, in their bones.

Resort to the hot bath would naturally be suggested when the pains are general, but it is contra-indicated by the already relaxed condition of the vessels. In several of Jaminet's earlier cases paralysis came on while the patient was in the bath, so that he was obliged to abandon its use. When the pain is local, however, hot applications limited to the affected part sometimes give temporary relief.

Frictions with or without stimulating liniments are generally employed; but the momentary relief afforded is probably obtained rather by diverting the attention of the patient than by any ameliorations brought about in the part.

Epigastric pain is generally relieved at once by the use of an alcoholic stimulant with ginger, as suggested by Jaminet.

Vomiting is to be treated with sinapisms to the epigastrium, and by swallowing bits of ice. If persistent it will yield to injections of morphine combined with atropine.

When paralysis occurs the treatment is to be conducted on general principles. Cups or leeches to the spine may be useful to some extent as revulsants, and to those may be added the alternate hot and cold spinal douche. In protracted cases, after the symptoms of congestion have subsided, strychnine may be useful. Electricity will aid in preserving the nutrition of the paralysed muscles. Constant watchfulness will be required lest the paralysis of the bladder be aggravated by over-distension. If the urine becomes ammoniacal and fœtid, great benefit will be derived from saccharine in doses of two grains three or four times a day. This will usually change the reaction of the urine, and prevent the fœtor.

With the exception of coma, the occasional cerebral symptoms are so transient as to call for no special treatment. Coma is an exceedingly grave symptom, from which recovery is extremely rare. The treatment will vary with the circumstances of the case. When the pulse is full and strong, venesection may be expedient.

But for the immediate effects of reduced pressure no treatment can compare with that originally suggested by Pol (20), and carried out to some extent by Foley, namely, the return of the patient at once into the compressed air. Foley says: "A true specific is returning to the caisson, through which means all such accidents (pains, vertigo, etc.) speedily disappear. It is to be resorted to unhesitatingly in all threatening cases, and the pressure should be admitted rapidly."

But the means of access to the caisson are not generally such as to promote readmission with the promptness required in a severe case. I therefore suggested, as the result of my experience at the Brooklyn Bridge, that a chamber should be constructed into which a cot containing the patient could be placed, and to which the compressed air could be admitted through a pipe connected with the caisson. After abatement of the symptoms, the pressure should be very slowly reduced, as much as several hours being occupied in the process.

As a temporary hospital at the works should be a part of any plant where compressed air is used, it would be easy to arrange a chamber of this kind in connection with the hospital. The chamber would resemble a horizontal boiler of convenient dimensions, having a door at the end, through which a cot containing the patient could be pushed in. It would be necessary, of course, to provide for a continual change of the air. Light could be admitted through bull's-eyes, and a system of signals arranged for communication between the patient and the attendant.

This suggestion was adopted subsequently at the Hudson River Tunnel, and found to be perfectly successful in every instance, a considerable number of threatening cases being completely relieved.

Of course it cannot be expected that secondary changes in the tissues will be relieved by simply reproducing the conditions which existed in the caisson. To be useful the treatment must be resorted to without delay.

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HÆMATOMYELIA

THE occurrence of primary hæmorrhage into the spinal cord is a condition which is not of frequent occurrence, and it is only of late years, by the careful observation and publication of cases with autopsy, that its existence has been fully demonstrated.

Charcot considered that all the cases were secondary to a primary myelitis; and Hayem, after a careful analysis of cases, came to the same conclusion: since that time, however, accumulated evidence has placed the occurrence of primary hæmorrhage into the spinal cord beyond doubt.

The rarity of spinal hæmorrhage, as compared with cerebral hæmorrhage, is accounted for by these facts:—first, that the vessels of the spinal cord, even if diseased, are not exposed to the same pressure as the cerebral vessels; and, secondly, that in the spinal form of the disease the greater proportion of the cases are due to trauma.

Etiology.—The large majority of the cases arise from injury—Oppenheimer says 90 per cent. Such injury may cause fracture, or dislocation of the vertebræ, together with hæmorrhage into the spinal cord; or it may give rise to hæmorrhage into the cord without any actual injury to the bony canal. In all of Thorburn's cases the hæmorrhage occurred at the level of the fourth, fifth, and sixth cervical vertebræ, and he considers it probable that the hæmorrhage is usually produced by a partial dislocation with recoil.

The mode of injury varies considerably; a fall on the back, a fall in the sitting position, a fall on to the feet, a direct blow on the back, forcible bending forward of the head as by a fall, the bearing a weight on the shoulders; any of these may give rise to this accident. Injury during labour has been shown by Schultze and Spencer to cause hæmorrhage into the cord of the child.

Excessive muscular exertion, prolonged exposure to cold (Benda, Boinet), suppression of menses (Eichhorst), and disease of the vessels are the assigned causes; but in a certain number of cases no definite cause can be found.

Hæmorrhage may occur secondarily to myelitis, from whatever cause arising, into tumours, and into pre-existing cavities in the cord.

Pregnancy, puerperal period, venereal excess, and hæmophilia may be named as remoter or contingent causes.

Age.—The disease may occur almost at any age, and cases are reported in a child at birth, in a child eleven months old, in a child four years old; but the majority of the cases occur between the ages of ten and forty.

Sex.—The male sex is much more frequently affected than the female. If, however, the cases due to injury be excluded, we find that it occurs almost as frequently in females as in males.

Pathology.—Hæmorrhage into the cord may be found under the following conditions:—

(i.) Capillary hæmorrhages, also called "accessory," are found scattered throughout the cord in cases where death has taken place from asphyxia, tetanus, and infective processes; these, however, give rise to no symptoms, and probably do not occur till shortly before death.

(ii.) Hæmorrhage occurring in myelitis—(*a*) a diffuse hæmorrhagic condition; (*b*) a focal hæmorrhage. The diffuse hæmorrhage is almost certainly due to a primary myelitis; but in the case of a focal hæmorrhage it is difficult, and sometimes impossible, to decide which is the primary condition.

(iii.) Hæmorrhage may occur into a tumour or pre-existing cavity in the spinal cord.

(iv.) Hæmorrhage may occur into a previously healthy cord in two forms—the first in which the hæmorrhage ploughs up the cord in a transverse direction and forms a round or oval-shaped hæmorrhage; the second in which the hæmorrhage, starting generally in the gray matter, but sometimes also in the white, ploughs up the gray matter in a longitudinal direction, forming an elongated hæmorrhage. The lines of least resistance to such a hæmorrhage would seem to be the gray matter of the posterior horns, so that in whatever portion of the gray matter the hæmorrhage starts it tends to limit itself to the posterior horns at the extremities. This is further borne out by the experiments of Goldscheider and Flatau, who injected the cords of dogs with Berlin blue in various situations and observed the course it took. Briefly stated, the results were as follows:—Injection into—(i.) the anterior horns, becomes at the extremities limited to the posterior horns and affects the white matter but slightly at the point of injection; (ii.) the lateral portion at the base of the posterior horn, limits itself almost entirely to the posterior horn, but affects the middle and anterior horns to a slight degree; (iii.) the posterior horns, limits itself to the posterior horns; (iv.) the middle zone, affects the gray matter of the same and opposite side, leaving the anterior horns less affected; (v.) the lateral horns, affects the white matter at the seat of puncture, the gray matter of the lateral horns, and the posterior horns; (vi.) the posterior columns, the injection remains limited to the posterior column white matter. Thorburn, however, says that it tends especially to affect the gray matter of the anterior horns and the central canal.

On opening the spinal canal, the spinal cord may present no abnormal appearance; in other cases the cord may be expanded at the seat of the hæmorrhage, and the dark colour of the blood may be apparent through the distended cord substance; it rarely happens that the hæmorrhage breaks through the membranes.

On transverse section of the cord a hæmorrhage is seen which usually occupies the central part of the cord, around which the cord is softened and of a yellow colour; unless the hæmorrhage be quite recent when the surrounding tissue is white.

In the later stages of the disease, if the hæmorrhage has been quite small, all that may be found is a small pigmented scar containing blood pigment crystals. In the case of more extensive hæmorrhage the result may be the formation of a cyst, and there is the possibility in still more extensive cases that the process of recovery may give rise to a syringomyelia or to a central gliomatosis; this view of the formation of such cavities is held by Langhans, Minor, Schlesinger, and Schultze.

Syphilitic disease of the blood-vessels has been found, and, in a case published by Williamson its rupture was the cause of hæmorrhage into the cord. Miliary aneurysms have been found in the cord by Griesinger, Liouville, and Hebold, but there is no evidence to show that they have ever given rise to hæmorrhage into the cord.

Symptoms.—The most characteristic feature of the disease is its sudden onset with loss of power in the limbs; it is usually attended by severe *pain*, its situation depending on the level of the lesion. In some of the reported cases prodroma have been present—numbness and pain in the limbs—some time before the loss of power. When the lesion occurs in the upper dorsal and lower cervical regions there is a sharp shooting pain passing through the chest and down the arms, the pain being usually localised to the seat of the hæmorrhage, and not extending along the whole spine as in meningeal hæmorrhage.

Motion.—Loss of power occurs rapidly in one or both arms, together with loss of power in the legs; the muscles are flaccid, there is retention of urine, loss of knee-jerks and of the superficial reflexes generally. Consciousness is usually retained, though occasionally it has been lost for a few minutes after the onset or injury. When the lesion is in the cervical region, myosis of one or both pupils may be present, drooping of the eyelid, and slightly raised temperature on one side of the face as compared with the other. Paralysis of the intercostal muscles will render the respiration entirely diaphragmatic. The pulse may be slow owing to the interruption of accelerator fibres in the cord.

Sensation.—Complete anæsthesia to all forms of sensation may exist below the level of the lesion, with a band of hyperæsthesia at the level of the lesion; this is said not to be present in some cases recorded by Parkin, and he also notes the fact that in testing the sensation from below upward or from above downwards there is a difference of an interspace in the result. The dissociation of the various forms of sensation may be a marked feature, in some cases, tactile sensibility remaining normal, while sensation to heat, cold, and pain is abolished either on both sides below the lesion, or on the opposite side to the paralysed limb, giving rise to a paralysis of the Brown-Séquard type. Minor has published eight such cases, with an autopsy in one case, and in this the lesion was at the level of the first lumbar vertebra, and the hæmorrhage extended from the conus below to the level of the exit of the spinal accessory above, and affected the left posterior horn and the hinder part of the anterior horn and the commissure. Similar cases have been published by Ross, Lloyd, Thorburn, and others.

Reflexes.—It is generally accepted that the condition of the reflexes depends on the seat of the lesion; certainly in a large number of cases the knee-jerks are abolished immediately after onset, whether the lesion be in the lumbar, dorsal, or cervical region; and it is also certain that in many of the cases the knee-jerks return and become exaggerated, together with spastic rigidity of the legs: on the other hand, there are cases in which the knee-jerks have remained absolutely abolished after an extensive hæmorrhage into the gray matter of the cord in the lower cervical region (Thorburn). That the absence or presence of the knee-jerks goes with the preservation or loss of pain sensation, as suggested by Bastian, would seem not to be the case invariably; for in the first case published by Minor there was complete thermo-anæsthesia below the second rib, with flaccid paralysis of the legs, yet the knee-jerks were increased; there was no atrophy of the muscles. The superficial reflexes are often abolished early in the disease, and return and become exaggerated later on.

Visceral symptoms.—Constipation and retention of urine are the rule during the earlier period of the disease, and give place to incontinence of urine and loss of control over fæcal evacuations in the later stages. Priapism is sometimes present, but its absence is frequently noted. Acute abdominal distension may occur.

The *temperature* is not generally raised at the onset of the illness, but rises slightly a few days after the onset; on the other hand, when the lesion is in the cervical region, the temperature may be abnormally low, as in a case recorded by Parkin in which the temperature fell to 77·6° F. at the time of death, and to 82° F. four days before death. The phenomena depending on a cervical lesion and also the occurrence of acute abdominal distension will be found fully discussed in a "Neurological Fragment," by Dr. Hughlings Jackson.

Late symptoms.—If the patient survive the acute stage of the disease, atrophy of the muscles in the limb or limbs affected, corresponding to the seat of the lesion, takes place. Electrical changes in the direction of the reaction of degeneration manifest themselves. Vaso-motor and sensory disturbances are present, the latter taking the form of preservation of tactile sensation and the abolition of the sensibility to heat, cold, and pain. If the lesion has been in the dorsal or cervical region, considerable loss of power in the legs may remain, with spastic rigidity, increased knee-jerks and ankle clonus; but without electrical changes, and without marked muscular atrophy.

Course and prognosis.—The disease may be rapidly fatal, owing to the extension of the hæmorrhage in an upward direction; or it may be fatal owing to extension by secondary inflammation occurring two to three days after the actual onset; or to the supervention of bronchitis or pneumonia, the intercostal muscles being paralysed. The patient may, however, recover from the immediate effects of the disease, and succumb to a purulent cystitis or to infection through a bedsore. In the more favourable cases partial recovery takes place, atrophy of some muscles generally remains, and the rigidity of the legs persists, with increase of knee-jerks and some difficulty in passing urine. The sensory disturbance, and especially the loss of sensibility to heat, cold, and pain, may persist for a long time, if not permanently.

Diagnosis.—(*a*) From acute myelitis it is at times impossible to separate either primary hæmorrhage into the cord or hæmorrhage secondary to the acute myelitis. In most cases the onset of myelitis is more gradual, attended by more marked prodroma, by a raised temperature and the gradual extension of the symptoms. That the onset in myelitis may be absolutely sudden is shown by a case reported by Williamson; pain was, indeed, absent at the onset, but according to his statement Leyden has shown that it may be absent also in cases of spinal hæmorrhage.

(*b*) From hæmorrhage into the spinal membranes four points may aid in the diagnosis—first, the marked irritation of the nerve-roots, as shown by the violent pains in the limbs and the jerking of the muscles; secondly (if the lesion be in the cervical region), the paralysis of the upper limbs is greater than the lower; thirdly, there is no myosis of the pupil which occurs when the hæmorrhage is into the cord; and fourthly, the dissociated tactile and temperature sense is not present.

(*c*) The disease is apt to be mistaken for acute poliomyelitis, especially in a child. Such a case occurred in a child four years old under the care of Dr. Cheadle, and another is reported by Prof. Clifford Allbutt in a child aged eleven months. [*Vide* art. "Acute Poliomyelitis," p. 186.]

(*d*) From syringomyelia and central gliomatosis it may be impossible to distinguish the later results of hæmatomyelia; one point brought forward by Remak may be mentioned, namely, that in gliomatosis the atrophy and the dissociated sensation occur in the same limb, whereas in hæmorrhage the limb on the opposite side to the atrophy is affected with regard to sensation. No proof of the above statement is given, the explanation is not obvious, and it needs further confirmation.

Treatment consists in absolute rest, the patient being placed in the prone position, with the application of an ice-bag over the seat of the hæmorrhage. The bowels should be freely open, and the retention of urine relieved by the use of the catheter. Ergot should be given by the mouth or subcutaneously. Later the treatment resolves itself into that of the bronchitis and pneumonia, and the prevention of cystitis and bedsores. The after treatment consists in the application of electricity and massage to the affected muscles.

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LIMITED DISEASES OF SPINAL CORD

I. SCLEROSES

DISSEMINATE SCLEROSIS[1]

SYN. :—*Multiple sclerosis*, *Insular sclerosis*, *Sclérose en plaques disséminées*, *Herdsklerose*, *Multilocular sclerosis*, *Polynesic sclerosis*.

DISSEMINATE sclerosis is a disease of the brain, spinal cord, and, it may be, peripheral nerves; characterised by areas of sclerosis of varying sizes, distributed at random, and destroying and replacing the nerve elements.

The first record we have of this curious disease is in the drawings by Cruveilhier in his *Anatomie Pathologique*, published between 1835 and 1842. The condition is depicted as it was seen in the pons medulla and spinal cord in four patients, and was called "gray degeneration." Carswell's *Illustrations of the Elementary Forms of Disease*, published in 1838, also contains pictorial representations of the morbid appearances.

The first clinical study of the disease was made by Frerichs in 1849, and some of his cases were subsequently examined after death by Valentiner, in 1856. It was not until six years after this that the condition was made the subject of careful study at the Salpêtrière, when Charcot, working under Vulpian, took the chief part in the investigations, and, in 1866, published a lecture on the subject which led to the disease becoming widely known. This production of the great French neurologist still remains a faithful picture of the classical type of the clinical manifestations of the disease; although we have since learnt that other types, equally characteristic but differing widely from that originally described, may be constructed.

In the interval which elapsed between the appearance of the publications of Frerichs and Valentiner on the one hand, and that of Charcot on the other, Turck studied the disease in its physiological bearings, while Rokitansky and Rindfleisch did much to advance the study of its morbid anatomy; so Baerwinkel too pointed out that the tremor was evoked on voluntary movement only.

After Charcot's publication, Leo and others studied the disease in

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[1] It would be better to use the name Insular sclerosis; for, even docked of a syllable, Disseminate sclerosis is too long.—ED.

Germany; but it was not until 1873 that any attention was given to the subject in this country, when Dr. Moxon recorded a case. Two years later the same observer published a paper dealing with eight cases (20).

Causation.—The cause of disseminate sclerosis is still shrouded in great obscurity, but much valuable information bearing on the subject has accumulated during recent years.

Heredity.—It has only happened exceptionally that any direct hereditary connection has been traced, or that more than one member of the same family has been affected. A case in point has been recorded by Leuch, in which a mother, the subject of disseminate sclerosis when pregnant, bore a child which remained quite well until the age of seven years, when the symptoms of disseminate sclerosis were noted; the child eventually died of this disease.

A still more striking instance of hereditary transmission has been recorded by Eichhorst, in which a mother and her son, aged eight years, were both the subjects of this disease; the author was able in both cases to verify his diagnosis by necropsy.

It more commonly happens that an indirect neuropathic inheritance can be established in the way of some other chronic disease of the nervous system; such as paralysis of some kind, epilepsy, or insanity.

Age.—Most commonly the symptoms become manifest in early adult life. Marie, who regards this as important in diagnosis, states that most cases occur between the ages of twenty and thirty. That the disease may occur in childhood seems proved, though Strümpell doubts the genuineness of most of such recorded cases. Meyer collected nineteen cases in 1887, Totzke collected thirty-four, and according to Pritchard more than fifty cases have been reported. Of thirty-one of Totzke's cases, in two the symptoms appear to have been present at birth; Ross stated indeed that the disease might affect children at the breast. In nineteen of Totzke's cases the symptoms were observed before the age of six years, and in twelve between the ages of six and fourteen years. As the clinical course of the disease is, as a rule, protracted, it is not surprising that the opportunities of necropsy in children have been few. The three cases with necropsies recorded by Zenker, Schüle, and Pollák, and regarded by earlier writers as proving that disseminate sclerosis occurs in children, were not instances of this disease at all. Its occurrence in childhood has, however, been established by competent observers; moreover, cases have been met with in adults where it has been possible to trace the origin of the disease to childhood. The fact that the affection may be met with in quite young babies naturally suggests the possibility of a congenital factor in its etiology. Of this we have no other evidence except that supplied by the morbid appearances met with, which have been likened by some observers to the gliomatosis found in cases of syringomyelia, and which have accordingly suggested that the starting-point of the morbid process is some congenital imperfection in the development of the neuroglial tissue. Two cases alluded to by Strümpell, in which disseminate sclerosis was accompanied by central gliosis and hydromyelia, are very suggestive in this connection. The association may have been a coincidence, but Strümpell asks with much reason whether such cases may not be an indication that disseminate sclerosis is a disease of endogenous origin analogous to syringomyelia, Friedreich's disease, or a multiple gliosis depending on certain anomalies of development comparable to multiple neuromas, fibromas, lipomas, and the like. Such a view would not exclude the possibility of other factors, such as infections, intoxications, traumatisms, and so on, intervening as occasional or additional causes.

It is rare for the disease to manifest itself for the first time after the age of forty or forty-five; though an instance of its occurrence at the age of sixty is on record.

Sex.—The affection is met with as frequently in the one sex as in the other; the statistics of some authors suggest a preponderance in females, of others a preponderance in males. Notably is the former the case in thirty-four instances collected by Charcot, in which twenty-four females were affected and only nine males. Ross, again, was of opinion that girls are more frequently affected than boys. The reverse, however, obtains in twenty-nine cases recorded by Blumreich and Jacoby, twenty-three of which were male and six female; while of fifty-eight cases collected by Probst, thirty-four were men and twenty-four women. So, too, in the statistics of Marie, Krafft-Ebing, and Redlich, more men were affected than women.

Occupation.—Oppenheim has insisted on the importance of ascertaining the patient's occupation in all cases of disseminate sclerosis; and has brought forward evidence to show that persons engaged in certain trades are liable to become the subjects of this disease. In all such cases some toxic agent is held responsible, notably prolonged impregnation with some metallic poison.

Intoxications.—As I have said, attention has been called to the importance of various forms of intoxication by Oppenheim, who found such causes operative in eleven out of twenty-eight cases in which the history was carefully inquired into. In most of the cases lead was the poison; but in some copper, zinc, and other poisons were operative. On the other hand, the clinical picture of disseminate sclerosis may be present during life but the characteristic lesions wanting on autopsy; this was the case in a patient of Oppenheim's, who suffered repeatedly from lead poisoning, and in whom combined system degeneration was found after death. Important as may be the immediate or remote influence of such toxic agents in certain cases, yet when we consider how many people, notably young ladies whose social surroundings are such as to exclude the possibility of their being exposed to any of these baneful influences, and who nevertheless become the subjects of disseminate sclerosis, we are forced to the conclusion that there must be some more general etiological factor about which at present we know but little. Alcohol is another toxic agent which has been supposed to account for the occurrence of some cases.

Infectious diseases.—Closely allied to the cases just considered are those which occur after one or other of the infectious fevers. Charcot refers to cases in which the clinical manifestations of disseminate sclerosis were first observed during convalescence from enteric fever, cholera, and variola. Kahler and others have regarded scarlet fever as especially apt to be attended with this result; and Marie found that enteric fever preceded the manifestation of symptoms of disseminate sclerosis in eleven out of twenty-five cases in which there had been an antecedent history of infectious diseases. Besides the conditions already noted, many other diseases have, more rarely, been the precursors of this affection—notably malaria; influenza, pneumonia, measles, diphtheria, whooping-cough, erysipelas, and dysentery. With regard to malaria much caution is needed, for what has been described in this connection by some observers as disseminate sclerosis is open to serious question. The nervous affection is said to occur under three conditions: temporarily in severe attacks of malarial fever; of variable duration after the fever; or suddenly with fever as acute attacks of malaria: the characteristic of all these conditions, in marked contrast to true disseminate sclerosis, being their tendency to result in recovery. Cases of the kind have been recorded by Torti and Angelini, Bastianelli, Camellis and Boinet, and Salebert; and the two first-named have regarded the symptoms as the result of an intoxication of the nerve-centres. That their explanation is the correct one is highly probable, and that many of the symptoms produced by this intoxication resemble those of disseminate sclerosis seems no less clear; but that these manifestations indicate the existence of the disease itself is an assumption which has no justification.

Caution is also needed in ascribing disseminate sclerosis to influenza, for, with influenza as prevalent as it has been of late years, it is not surprising that the association of the two diseases should have been met with in a few instances. The true explanation of their association may, however, be that disseminate sclerosis, previously present in slight degree and unrecognised, may become much more evident in the debilitated state following influenza, and the disease may then make much more rapid progress.

Probst found joint rheumatism an antecedent in five out of fifty-eight cases, and in one case of disseminate sclerosis the patient became much worse after an attack of rheumatism; as did another after an attack of pneumonia.

Strümpell does not regard the infectious hypothesis as admissible, for in the last twenty-four cases of disseminate sclerosis that he has seen he has not been able to establish any such connection. Krafft-Ebing could only establish a relationship of the kind in six cases out of fifty-three.

Moncorvo more especially has attempted to prove an etiological connection between syphilis and disseminate sclerosis; Michailow and Jacobsohn also regard syphilis as a cause.

Pregnancy.—Sir William Gowers has met with cases in which the disease began during pregnancy, remained stationary until the next pregnancy, and then became progressive. Pregnancy and parturition certainly have a deleterious effect in cases of disseminate sclerosis, and are responsible for relapses or more rapid progress of the disease.

Antecedent affections of the nervous system.—When symptoms of disseminate sclerosis have been related to any preceding affection of the nervous system, they have usually developed gradually a year or more after the manifestations of the antecedent disease have disappeared. In some instances certain phenomena persist which are attributable to the primary disease, but these may be stationary. Some form of myelitis, either primary or following an injury, has been most commonly met with in this association. According to Leyden, cases may begin like an acute myelitis and go on to sclerosis. Disseminate sclerosis may be found after death in cases in which, many years before, some acute illness had been followed by disseminate myelitis.

Trauma.—Apart from any indirect influence, through the production of myelitis, trauma appears to play some part in producing the disease; but whether a fall or blow is alone capable of generating such a process, or whether it is only the means of lighting into activity some dormant pre-existing morbid condition, congenital or otherwise, must remain for future research to determine. Mendel has more especially insisted that the disease may be caused by injury; and Blumreich and Jacoby established a history of injury in eleven of their twenty-nine cases; but in only five of these was the connection sufficiently close to be of value.

Cold.—Exposure to cold or chill has preceded the occurrence of a good many cases; thus, in a case observed by Baerwinkel, the patient fell into water and allowed his clothes to dry on him; three days later symptoms of disseminate sclerosis showed themselves. Some reserve is needed in accepting such cases—as careful inquiry will sometimes elicit evidence that the disease was in existence prior to the wetting or exposure; though the symptoms had not been sufficient to arrest the patient's attention. In no fewer than forty out of his fifty-three cases, however, was Krafft-Ebing able to obtain a history of severe chill as an antecedent; and Probst concludes that chill is the most common cause.

Fatigue.—What has just been said with regard to wet and cold applies equally to fatigue; the probabilities being that where over-exertion has appeared responsible for the disease, the true explanation was, that the fatigue merely accentuated or evoked phenomena previously existing, though more or less dormant.

Emotion.—It is difficult to see how emotional influences can play any part in the generation of an affection like disseminate sclerosis, in which such pronounced structural alterations occur in the nervous system; nevertheless, the association occurs; as in a case of Suckling's, recorded by Jordan, in which the symptoms of disseminate sclerosis appeared suddenly a few days after a severe mental shock. It is probable that the explanation suggested for the cases following chill, fatigue, or injury, is equally true in the class of cases now under consideration; and certainly any of these factors seem adequate to determine relapses, or to accelerate the morbid condition otherwise originated.

Symptoms.—Three types of the disease are commonly described, a cerebro-spinal, cerebral, and spinal; according as the morbid process affects both or one or other of these parts. In the majority of instances, however, so far as the central nervous system is concerned, the disease is general; and, while the earlier manifestations may be of cerebral or spinal origin, unmistakable evidence of affection of both of these parts appears sooner or later. This does not mean that in one class of cases the cerebral symptoms may not preponderate, or that in another the spinal symptoms may not be in the ascendency; what I wish to insist on is that the disease, as a rule, affects the whole of the central nervous system; and that any attempt to distinguish a cerebral from a spinal type on the ground that one or other part of the nervous system is affected to the exclusion of the other, is as a rule unwarranted. It is exceedingly rare for the disease to be limited to the brain; and even older writers, such as Ross, admitted that, though psychical disturbances predominate in the cerebral form, the course of the disease does not otherwise differ materially from the cerebro-spinal variety. In some cases the disease, throughout its clinical course, manifests itself by spinal symptoms only—there being an absence of cerebral symptoms, such as psychical disturbances, apoplectiform attacks, vertigo, nystagmus, and intention tremor; but we are now fully alive to the fact that sclerotic patches may be met with on necropsy scattered throughout the whole extent of the cerebro-spinal axis, and yet their distribution may have been such as to give rise to the clinical phenomena of a spinal disease only. Exceptionally, the patches of sclerosis have been found limited to the spinal cord; in this respect, however, we can rely only on the more recent observations; for, with much less perfect methods of investigating the morbid changes in the nervous system than those now in vogue, areas of sclerosis, possibly ill defined, might easily have escaped detection formerly which would now almost certainly be recognised by a skilled observer. So, too, the length of time during which the patient manifested the disease before death is another important consideration in this respect; for it is conceivable that a disease, the course of which is ordinarily very protracted, might result in death before the morbid process had had time to make incursions from its original site into other parts. It appears best then to regard disseminate sclerosis as a disease which affects the whole central nerve axis, in unequal degree, it may be, in different parts, or earlier in some parts than in others; not as an affection in which any hard and fast line can be drawn between a form limited to the brain and one limited to the spinal cord.

The degree in which nerve-roots and peripheral nerves, both cranial and spinal, are involved varies greatly in different cases; but there is unquestionable evidence of their implication irrespective of affection of their nuclei of origin.

Far more important than any division of the disease into the three types that have just been discussed is the recognition that its clinical manifestations may differ widely from those commonly regarded as characteristic; in aberrant cases few if any of the classical symptoms by which the disease first came to be recognised may be present. Dr. Buzzard, who has made this disease the subject of long and careful study, and who has done much to elucidate many of the intricate problems connected with its clinical manifestations, has insisted on the extreme importance of recognising that such irregular instances of the affection occur; albeit most modern observers are agreed that from the cases formerly regarded as aberrant, another type of the disease may be constructed to which cases may be referred as commonly and as definitely as the former kind of cases may be referred to the classical types. The importance of this observation is accentuated by the fact that many of the manifestations of the new type, to which attention is now being called, are only to be distinguished from hysteria with great difficulty. The gravity of an error of diagnosis, which would subject a patient with so serious a form of organic disease to the rigid system of discipline so necessary in the treatment of hysteria, is too obvious to make it necessary for me to dwell on this subject at greater length.

After what has already been said, I make no apology for departing from the usual custom of describing first the clinical pictures of disseminate sclerosis, as they are to be found in the classical writings of Charcot and others. We are all impressed strongly enough with these pictures, which most of us have learnt to recognise and interpret aright. What appears to be wanted at the present time is a recognition of the multiformity and frequent latency of the disease. It must not be supposed, however, that the aberrant cases escaped the observation of Charcot and others of the older writers on this subject; for, since the discovery of the anatomical lesions of disseminate sclerosis, by Pitres in 1877, in two cases otherwise diagnosed during life, such anomalous cases ("formes frustes") have been recognised; yet it is only within recent years that the frequency of their occurrence has been appreciated.

As has already been said, such cases are too commonly regarded as "hysterical"; and such an error of diagnosis may be perpetuated for years. Women are more frequently affected than men, though cases of the kind occur in the latter sex also. It commonly happens that the first manifestations occur after some mental or physical shock. These may be but little observed at first; a peculiarity in the behaviour of the patient is readily ascribed to hysteria: for instance, there may be temporary aphonia; or sight might be impaired or lost for a time, and then recovered—a not uncommon mode of onset in these cases. Or it may be that a patch of numbness is noticed somewhere, perhaps so slight and so fleeting that it is only recalled to the patient's memory on close inquiry into the earliest manifestations of the disease. More rarely, subjective sensations of "deadness," "coldness," or "pins and needles" are first complained of. But perhaps the most common phenomenon noted in the early stages is loss of power in one or more limbs, usually evanescent;

less frequently the earliest manifestations consist rather in over-action, and thus convulsions may occur.

However this may be, the all-important fact to be borne in mind in this type of the disease is that such initial symptoms may entirely pass off, to return with like abruptness at some subsequent period; or again, without entirely clearing up, they may diminish and remain in abeyance, it may be for years. It is no uncommon experience to hear that a patient presenting one or other of the above symptoms has quite recovered; but a few careful questions concerning the previous symptoms will elicit evidence that the disease, supposed to be cured, is only temporarily dormant. You hear that the patient, as a rule, walks quite well; but that when she gets rather tired she limps a little; or there is occasional double vision; or sight is not quite good at times, or the patient is sometimes rather hysterical in manner. Such symptoms are of grave import; and, to the experienced, indicate with certainty that there is but a temporary lull, and that at some future time, near or remote, the disease will once more assert itself with fresh vigour.

The physician who takes a grave view of such cases at the onset is for a time, for years it may be, regarded as having made a mistake in diagnosis; while those who regard the case as purely "functional" or "hysterical" are lauded for their acumen. But those experienced in such cases can afford to allow others whose views differ from theirs to have this brief period of triumph; for they know only too well, unfortunately for the patient, that the time must come when the truth of their original opinion will become only too painfully evident. After an absence, perhaps even for years, symptoms return, without obvious cause, during pregnancy or after parturition; under some debilitating influence, as of an acute illness of some kind; on some injury, exposure to cold, privation, or mental or physical shock. The original symptoms may return with greater intensity, or new symptoms may manifest themselves. Paretic weakness may return in a limb previously thus affected; or may disable some other limb, alone or in conjunction with that first attacked.

Again the symptoms may clear up, partially or almost entirely, to return once more at some subsequent period; or they may remain and continue to progress. But even when the symptoms thus persist, and even progress, this remarkable periodicity of the disease still manifests itself from time to time; transient improvements recur for a while, and false hopes are awakened.

Once fully established, the disease progresses with unerring certainty to death; though this may be long postponed, and there may be many periods of remission before the end is reached. Yet even at this advanced stage of the disease many of the classical symptoms of disseminate sclerosis are wanting; and, in their absence, the inexperienced, while no longer doubting the organic nature of the disease, find it difficult to reconcile the picture before them with that of classical disseminate sclerosis, and with their own experience of classical cases.

On the other hand, such a patient may be quite bedridden with complete paraplegia, with or without spasticity and contracture; and the upper limbs, and even the neck muscles, may share in the loss of power. Groups of the affected muscles may waste. In areas of numbness there may be blunting of sensibility. Any movement may induce a sensation of giddiness. Sight may be extremely impaired, with or without evidence of a form of optic nerve atrophy to be subsequently described as characteristic of this disease. The tendon reflexes may be exaggerated, and there may be ankle clonus; more rarely the knee-jerks are abolished. The power over the sphincters may be impaired or completely lost. Such a patient may remain perfectly clear in intellect, but at times emotional; or, on the other hand, he may pass into dementia and fatuity.

As advanced a stage of the disease as this may be reached, and even death may supervene, without nystagmus, intention tremor, or a scanning utterance—features so prominent in the classical disease.

So varied are the clinical manifestations of disseminate sclerosis that indeed many types of the disease might be constructed in addition to the three mentioned at the outset of this article, and that which up to this point has engaged our attention. A brief allusion will be made to one or two of these after we have considered the classical types.

The disease, as portrayed by Charcot and most subsequent authors, is a well-marked and distinctive one, not easily confounded with that of any other affection; and even at the present time, to the majority of medical men, the mental picture which is at once called up by the name is one differing widely from that which has just been described. Until within recent years the most common group of symptoms has been conceived as follows:—The patient slowly falls into a spastic paraplegia with exaggerated knee-jerks, and, it may be, ankle clonus. No real motor weakness of the arms occurs, but on any attempts at movement a curious intention tremor of an irregular, jerky character is evoked. Closely allied to this is the nystagmus; which is either spontaneous in character or evoked only on lateral, on upward, or more rarely downward displacement of the globes. Diplopia, with paresis of some external ocular muscle, is among the paretic symptoms recognised as of no infrequent occurrence. An integral part of this group of symptoms is a peculiar defect of speech, described as a scanning utterance, in which the pauses between words, and it may be syllables, are increased with undue accentuation of the latter, and a tendency to elide the ends of words. Apart from the occurrence of paræsthesiæ, in the form of numbness, coldness, pins and needles, and the like, sensory defects are rare. Amaurotic defects of vision may occur; there may be tinnitus aurium, and vertigo is common. Psychical disturbances occur in which the mental faculties become blunted; or there may be a manner suggesting a state of well-being quite out of keeping with the patient's physical condition. In a small percentage of the cases apoplectiform or epileptiform seizures are met with. Trophic disturbances, muscular or cutaneous, are among the possible late manifestations of the disease; and the sphincters remain intact until the same stage, when, in the event of control being lost over the sphincter of the bladder, cystitis and pyelitis result as final complications.

Or secondly, the clinical manifestations throughout may be those of a spinal cord affection; the psychical disturbances, vertigo, apoplectiform attacks, optic nerve atrophy, nystagmus, scanning speech, intention tremor, and so on, being absent. There is always some form of motor disturbance in the legs, the amount of impairment of power varying in different cases. The defect is usually of the spastic type, with exaggerated knee-jerks, and perhaps ankle clonus; but sometimes there is an absence of spasticity, and not only may the knee-jerks not be increased, but they may be actually abolished; in the latter case the knee-jerks commonly disappear and reappear at intervals. There may be unsteadiness, titubation as in cerebellar disease, or actual ataxy. Various paræsthesiæ may be complained of; and sometimes these are accompanied by actual blunting or loss of sensibility, of variable distribution. There may be some difficulty in passing water early in the course of such cases, or there may be some loss of control over one or both sphincters; though usually such defects are among the later manifestations. If the morbid process invade and destroy the anterior horn cells of the lumbar enlargement, groups of muscles may become atrophied. Trophic disturbances of the skin, in the form of bedsores, belong to the later stage, and are associated with loss of control over the sphincters. From the above description it will be manifest that this type of the disease is easily confounded with primary lateral sclerosis, amyotrophic lateral sclerosis, ataxic paraplegia, and tabes dorsalis. The points on which we have to rely for diagnosis will be discussed subsequently.

Then again hemiplegia may be the predominant symptom, though this variety is not common, and the hemiplegia is as a rule transitory.

The rarest form of the disease is that which simulates amyotrophic lateral sclerosis in its clinical manifestations. Cases of the kind have been recorded by Pitres, Déjerine, Skolosubow, and Probst.

It can serve no useful purpose to multiply clinical types of this disease unnecessarily; but one other deserves brief notice owing to the frequency of the cases. Here the symptoms all point to a spinal cord affection, with the exception of the peculiar form of optic atrophy, which probably affects chiefly or solely the temporal halves of the discs. Some such cases—where the knee-jerks are absent, and shooting pains, ataxy and Romberg's sign are present without psychical disturbance, scanning speech, nystagmus, or intention tremor—may be mistaken for cases of tabes; but there is usually sufficient evidence of loss of motor power without muscular atrophy to prevent this error.

Finally, we must remember that cases often fall away from the spinal type; that, as Charcot insisted as long ago as 1876, paraplegia may be an almost isolated symptom, to which, however, may be added some of the other signs of the disease; and that it is exceptional not to find two or three of the characteristic associated phenomena in some stage of the disease.

It is scarcely necessary, or even desirable, to construct a type for the third or mixed form of the disease; for, as I have said, the disease is cerebro-spinal in its distribution, and sooner or later will present symptoms of a mixed character accordingly.

So manifold are the phenomena of disseminate sclerosis that it behoves us to consider in detail the various symptoms that may arise; and after the general survey of the subject which we have already taken, this may now be done with advantage.

Motor disorders.—One of the most characteristic features of the ordinary form of the disease is a peculiar irregular jerky tremor which only occurs on attempts at movement, and ceases as soon as the muscles are at rest again. This symptom was present in seventy-five per cent of the cases collected by Probst. At the beginning of the act the tremor is, as a rule, slight; but it increases in range until, when the object aimed at is all but or actually attained, it reaches its height; at this stage the to-and-fro jerkings may be of the wildest possible character. The excursions are large and are slowly rhythmical. If such a patient attempt to raise a glass of water to his lips most of the contents will probably be spilt long ere the glass reaches its intended destination. The tremor is not limited to the muscles actually in action, but spreads to neighbouring ones; so that if the arm is being moved there may be not only tremor of it, but also of the head and upper part of the trunk.

While, as has already been said, these cases may be under observation for years without any tremor being detected, no part is exempt from it on attempts at movement. It may be seen in the facial muscles, in the ocular muscles as nystagmus, and laryngoscopic examination may reveal similar tremor of the vocal cords on phonation—a condition of things which is also evidenced by a tremulousness of the voice. The tremor of the neck muscles causes oscillation of the head; and, as this goes on while the head appears to be at rest when the patient is sitting, this seems to form an exception to the rule that the tremor only occurs on movement; in reality the neck muscles are not at rest, for though the patient is sitting still, the neck muscles are constantly in a state of action as they support the head in the erect position. In such cases immediately the patient lies down the tremor ceases, to return, of course, on any attempt to sit up.

In no part of the body is the tremor more commonly present than in the arms; and a common method employed for evoking it consists in asking such a patient to touch the point of the nose with the tip of the index finger of the extended limb. In slight cases the unsteadiness may only be observed towards the completion of the act; but in severe cases it begins with the voluntary movement, and becomes of wide range by the time the tip of the nose is reached. The handwriting is altered in a characteristic manner; for the first words of a sentence may be readable, while, owing to the increasing unsteadiness of the hand, the sentence becomes more and more illegible as it proceeds. The trunk may oscillate when the patient sits or stands, and the respiratory muscles may be affected, resulting in jerky breathing.

When present in the legs the tremor is evoked by standing, or when the patient begins to walk; but it may also be brought out if, when the patient is in the recumbent posture, he be made to raise the leg off the bed to attempt to touch some object held above it.

The intention tremor may give rise to ataxic movements indistinguishable in themselves from those of true ataxy due to sensory defect; and the similarity is increased by the fact that in some cases the patient can stand if supported, but falls if left to himself. Both conditions are, however, readily distinguished from true ataxy as closure of the eyes does not in any way affect it. Strümpell, however, regards the intention tremor as an ataxic disturbance; and in a boy with tumour of the corpora quadrigemina, observed by Bruns, there was intention tremor of the arms and ataxy of the legs. So too Goldscheider has observed cases in which disseminated patches of sclerosis, especially in the pons, caused acute ataxy, which afterwards assumed such characters that it became impossible to distinguish it from intention tremor. No satisfactory explanation of the mode of origin of the intention tremor has yet been offered. Charcot's view was that the axis-cylinders, denuded of their myelin sheaths, offer different degrees of resistance to the passage of impulses at different points in their course; so that such impulses are variously retarded in different axis-cylinders, and, reaching the muscles in an irregular manner, thus evoke irregular contractions of them. This explanation is supported by two observations by Sir William Gowers in which, probably as a result of pressure on the motor path, intention tremor was present in a case of a tuberculous growth in the crus; and in another case of a similar growth in the pons.

It cannot be said that the experimental observations of Pasternatzky, who attempted to produce intention tremor in animals by lesions of the motor tracts, have helped us to any extent in this difficult problem. Erb is of opinion that the tremor depends on the precise position of the patches of sclerosis, where it disturbs some mechanism for the co-ordination of movement. In support of this view it has been said that the symptom is absent when the sclerosis is limited to the spinal cord, but occurs when the pons is affected. Stephan, on the other hand, regards the optic thalamus as the seat of lesion in such cases; according to Oppenheim, a similar form of tremor is met with in cases of cerebellar tumour. This I have seen; and, moreover, after ablation of the cerebellum in animals the oscillation of the head on any attempts at movement strikingly resembles that seen in disseminate sclerosis. Of all the possible explanations that have been offered of the way in which the intention tremor results, none appears to me so satisfactory as that which supposes it to be due to a failure of synergic action of muscles in purposive movements consequent on an unequal degree of innervation both of the muscles directly concerned and of their antagonists.

Apart from the irregular jerky movements on the performance of voluntary acts spontaneous movements sometimes occur over which the patient has no control; such as attacks of hiccough, yawning, laughing, crying; and the latter phenomena are, as a rule, unattended by the corresponding emotional feelings. Such conditions may be met with early in the course of the affection, and a slight degree of this state of things is not uncommon. In that lesions of the medulla and pons are so commonly associated with emotional phenomena, it is probable that the occurrence of such emotions in disseminate sclerosis is consequent on patches of sclerosis in these parts of the central nerve axis.

Rigidity.—Spasm of the muscles is one of the most frequent symptoms of the classical type of multiple sclerosis. Though the arms do not escape, the legs are more commonly affected, and in greater degree. Spasm in the extensors preponderates at first; but the flexors ultimately gain the ascendency and lead to permanent contracture, in which the legs may be so drawn up that the heels are pressed against the nates. Contracture of this kind in the arms is rarely seen. Even before contracture voluntary movements are hampered by the rigidity; and the latter increases with movement, so that it may ultimately prevent the possibility of further movements for a time. Passive movements produce the same result, the stiffness increasing until it may require a very great effort to overcome the resistance occasioned by the spasm. In some instances the spasm is limited to a single group of muscles, and the rigidity thus occasioned may pass on to contracture.

Tendon-jerks.—With this condition of rigidity the tendon-jerks are in excess of the normal; but it may so happen that the full effect of this increased irritability is masked, or altogether prevented, by the spasm actually preventing the responsive movement in the limb which would otherwise result from a tap on the tendon. The increased activity of the tendon-jerks is manifested by an excessive jaw-jerk, amounting even to clonus; and by excessive arm-jerks and knee-jerks, with, it may be, rectus clonus and ankle clonus. On the other hand, in a small number of cases the knee-jerks are abolished.

Motor paralysis.—Some motor weakness is common to all the clinical types of disseminate sclerosis; but no symptom is more variable as regards its mode of onset, the degree of impairment, and its precise behaviour. These variations have been sufficiently insisted on when discussing the type of the disease so apt, more especially in its earlier stages, to be mistaken for the manifestations of hysteria. The motor weakness may or may not be associated with tremor or rigidity, but in either case the weakness rarely amounts to considerable paralysis. As a rule all movements can be executed, though feebly, slowly, and with difficulty; and, as fatigue is soon induced, the first movements attempted are always performed better than subsequent ones. Moreover, where there is actual rigidity, or even a subjective feeling of it, fatigue results the more readily in that there is a sense of resistance, true or supposed, of the spasm to be overcome. Though no part is exempt from loss of motor power, the legs are most commonly affected; the arms, however, suffer almost as frequently. In accordance with what has been already said when treating of these symptoms individually, such motor weakness is most commonly associated with rigidity in the legs, and with jerky tremor in the arms. The latter association also occurs in the neck muscles, when they are affected, and with the muscles of the eyes. It is probable that the peculiar defect of speech depends on the same association. We have seen that no part is exempt from motor weakness; so that, in addition to the parts already considered, paresis or paralysis may be met with in other regions, more especially in the territories dominated by other of the cranial nerves. Besides ocular paralysis, the motor division of the fifth cranial nerve may be affected, resulting in difficulty of mastication; thus the facial muscles may be palsied as a result of a lesion interrupting the motor path at some point on the cerebral side of the facial nucleus, with the result of isolated paralysis of the lower facial; or involving the nucleus or emergent fibres of the nerve, when the upper face is affected as well as the lower, and well-marked degenerative reaction is found in the affected muscles.

Sclerotic patches in the medulla may lead to difficulty of swallowing associated with paralysis of the palate, which may be unilateral or bilateral; and, alone or in conjunction with such defects, paralysis of the vocal cords may exist, when the abductors suffer alone or before the adductors. The tongue may show the paralytic deviation due to a lesion interrupting the motor path to it at some point on the cerebral side of the hypoglossal nucleus; or the lesion may be in the medulla affecting the nucleus or emergent fibres of the hypoglossal nerve, when atrophy on one or both sides of the tongue results, according as the lesion is unilateral or bilateral. In consequence of defects of the same kind in areas of sclerosis in the medulla various articulatory defects of speech are met with; alterations of tone of voice also, and, it may be, suffocative and other attacks which endanger life.

Before leaving the subject of motor paralysis, allusion must be made to curious apoplectiform attacks which sometimes occur, and which may give rise to hemiplegic weakness, indistinguishable from the result of rupture or occlusion of a cerebral artery, save by its fleeting character; in this it closely resembles the apoplectiform seizures which occur in general paralysis of the insane. A further resemblance to the attacks in general paralysis is seen in the tendency to repetition of these attacks, perhaps several times in the same subject. The hemiplegia, as a rule, is of the ordinary form, and may be accompanied by aphasia; or there may be alternate paralysis, the limbs being affected on one side and the territory presided over by some cranial nerve on the other.

Gait.—We are now in a position to study the various alterations in the patient's mode of progression. Paralysis, whether spastic or flaccid, may be in such degree as not only to forbid walking, but to exclude all possibility of standing; but when progression is still possible the most varied gaits are met with. The most common of these is a spastic paralytic gait in which the combination of weakness and rigidity hamper the movements. The inferior extremities are slightly flexed, and cling to each other and to the ground, so that they are unlocked with difficulty; and, as they are brought forward in turn, the anterior half of the foot scrapes along the floor. On the other hand, there may be a combination of spasticity and unsteadiness; so that, in addition to the clinging of the feet to the floor and the difficulty of moving the lower extremities, the patient stands on a wide base, and, as every movement is uncertain and reeling in character, he tends to fall. Closely similar to this state of things is the combination of spasticity, paresis, and ataxy, described by Oppenheim, in which the legs are stiff and held widely apart from each other; the movements are slow and the anterior half of the foot clings to the floor, but once it has been effectually released the limb is sharply raised in the air and the heel is stamped down. The same observer has noticed an ataxic condition in one leg and a combination of spasticity and paralysis in the other. Much more rarely a purely cerebellar gait is seen, in which there is unsteadiness, titubation, and reeling like a drunken person. Or the gait may be more or less ataxic in character. Apart from hemiplegic weakness of apoplectic origin, one leg may be affected alone; or one may be more affected than the other: thus all sorts of curious combinations are seen, giving rise to the most grotesque modes of progression. Rapid variations in the character of the gait and in the degree of defect occur in consequence of the sudden changes and variations so characteristic in all the symptoms of multiple sclerosis; and in many cases all that is observed in the earlier stages is a feeble gait having no peculiar characteristics other than those common to all conditions attended with feebleness of the lower extremities without spasticity. Patients are sometimes met with who, though unable to rise from the sitting to the erect posture without assistance, can nevertheless walk unaided, and without showing any peculiarity other than a feeble gait and a tendency to become soon fatigued.

Speech defects.—"Scanning," "staccato," or "syllabic" speech is one of the symptoms of the classical disease, and was supposed to be of frequent occurrence. In reality, however, many cases present no alteration in speech; and the majority of patients who do, only speak slowly without the scanning character being distinctly marked. When typically present it is very characteristic; the words are uttered slowly, the syllables, and more rarely the words, have the pauses between them lengthened, and each syllable is unduly accentuated, though there is a tendency to elide or slur over the ends of words. The speech may have a nasal character in consequence of paresis of the palate; but, apart from this, the pronunciation of certain letters is also somewhat indistinct, especially the voiced explosives such as *b*, *d*, *g*, and the extra fricatives *c* and *r*, which are to be produced by the second and third stop-positions described by Wyllie. The voice is monotonous, according to v. Leube, in consequence of the inability to alter quickly the height of tone and accentuation of syllables. Moreover, according to the same observer, such patients are unable to maintain any tone in its full intensity for any length of time; the note gets higher and higher, possibly from failure to regulate the tension of the vocal cords in response to the increasing strength of the expiratory blast. Occasionally there is aphonia; or speech may be interrupted from time to time by noisy inspiratory sounds consequent on incomplete opening of the glottis.

As in respect of other muscular movements, the first words of a sentence are often much more clearly spoken than those at the end, when the muscles of speech become fatigued. Indeed this abnormal tendency of the muscles to tire is one of the factors in the causation of the syllabic utterance. Not only may the muscles of the lips, tongue, palate, and larynx be at fault, but also those concerned with the respiratory movements. The researches of Goldscheider have shown that normally there is an increase of the expiratory blast at the beginning of each syllable; and this blast is so controlled in the mouth or throat as to be sufficient to complete the articulation: whereas in disseminate sclerosis he finds that the curve produced by the expiratory blast is not a steep but a flat one, and that variations in intensity of the expiratory stream of air can only be altered slowly. The same observer has shown that graphic records of expiratory pressure reveal that during a long sustained note the line of the normal curve is straight, while in multiple sclerosis it is wavy. The explanation which has been offered of this phenomenon is probably the correct one; namely, that it is dependent on oscillations of the vocal cords, a condition which may be seen to exist on laryngoscopic examination, and which v. Leube described long ago; this narrows and widens the glottis, and gives a tremulousness to the voice. Another interesting point, brought out by Goldscheider's investigations, is that in disseminate sclerosis the curve produced by vocalisation may suddenly rise abruptly, as the patient is unable to keep up uniform tension of the vocal cords for any length of time; the cords separate from each other, and thus more air suddenly escapes, causing the abrupt rise in the curve. He has also shown that a similar inability to control the tongue can be demonstrated by making the patient vocalise *r*; normally the line of the curve should show small regular oscillations, whereas in multiple sclerosis, owing to the slowness and difficulty with which the tongue can be brought into the proper position, the oscillations are absent in the first part of the curve, and in the latter part are imperfectly marked. The difficulty with explosives is also well shown by this graphic method; moreover, it can be seen that the distinctness of the pronunciation of one of these letters, and the steepness of the rising curve, depend on the time the patient has to prepare before uttering the sound. If another word immediately precede the explosive, and especially if another explosive precede it, the letter is indistinctly and softly pronounced, and the curve shows a flat gradual ascent.

True aphasic defects are very rare in multiple sclerosis.

Ocular phenomena.—So important are the ocular phenomena of disseminate sclerosis, more especially in their bearing on the difficult problem of diagnosis, that they call for our most careful consideration. Disturbances of vision commonly occur, and may or may not be accompanied by changes in the optic nerves, perceptible on ophthalmoscopic examination. Such patients usually complain of a mist before their eyes, rarely of specks and flashes. The defect in vision may be of very rapid onset, in some cases, indeed, quite sudden; and it may be the earliest manifestation of this mysterious disease: on the other hand, it may be of slow and gradual onset. Whatever the rate of progress, the same variability is met with in this symptom also; the most marked amblyopic disturbance may be followed by a period of remission in which vision is greatly improved (see art. "Medical Ophthalmology," vol. vi.), and then later the defect becomes as bad as ever again. Many such relapses and improvements may occur in the course of the disease before permanent amblyopia is established. It is common to meet with cases in which transitory defect of vision, never amounting to complete amblyopia, has existed in one eye, for, it may be, some months or a few weeks only, which has then cleared up, but is followed at some subsequent period by precisely the same defect in the opposite eye; or indeed in the eye originally affected.

Of the anomalies of the fields of vision central scotoma for colours is a prominent feature. Uhthoff, who has devoted much attention to the ocular defects in disseminate sclerosis, found central scotoma in fifteen out of twenty-four cases of this disease (see art. "Medical Ophthalmology"). Another alteration of the fields which occurs is that of peripheral limitation; and Uhthoff found this contraction of the fields in eleven out of twenty-four cases. Dr. Buzzard, though unable to give figures, would have expected a larger percentage of these defects as a result of his observations.

Both the transitory amblyopia and the contraction of the visual fields are symptoms which may readily be mistaken for indications of hysteria in a disease in which, as we have already seen, there are many manifestations indistinguishable from those of the functional disorder; it therefore becomes all-important that in any case in which these phenomena are met with, a careful search should be made by ophthalmoscopic examination for changes in the optic nerves. The most common change in the optic nerves is pallor, often so slight in the earlier stages as to be difficult of interpretation; at this time any inequality in the degree of affection of the two nerves may be of most important pathological significance. Though the occurrence of optic atrophy did not escape the observation of Charcot, and of others who have studied disseminate sclerosis, no one has made so exhaustive a study of this phase of the affection as Uhthoff. Among previous observers, however, Guanck's statistics, quoted by Ross, must be mentioned; for of fifty cases which he observed there were defects of vision in twenty-eight, and changes in the optic discs in fifteen; these defects were atrophic in character in all but three, in which there were hyperæmia and neuritis. Uhthoff found changes in the optic nerves in 45 per cent of a hundred cases of disseminate sclerosis which he examined ophthalmoscopically. In two of the cases there was marked atrophy of both nerves; in one a similar atrophy existed on one side with pallor of the temporal side of the opposite disc. In seven cases the whole of the disc was incompletely atrophic on both sides; in four this change was seen in one eye only, while the temporal side of the other disc was pale; in eight cases the change could be detected in one eye only. In eighteen cases the atrophy was partial, and was limited to the temporal half of the disc; and in five there was distinct evidence of optic neuritis—well marked in three, and slight in two.

Dr. Buzzard, who has also given a great deal of attention to this subject, has placed on record figures which closely agree with those of Uhthoff; out of a hundred cases of disseminate sclerosis, drawn from hospital and private practice, there was pallor of the discs in 43 per cent. The same observer has never met with anything that might strictly be characterised as optic neuritis or papillitis; though he has seen what he has described as a dark gray discoloration "somewhat resembling the tint of hyperæmic gray matter of the cerebrum." While this intense hyperæmia probably represents a stage in the process which subsequently results in atrophy, it is probably never present in the large majority of cases. When such a change is seen with the ophthalmoscope it depends no doubt on affection of the optic nerve, at its termination in the globe, by a patch of the same morbid change which occurs in other parts of the nervous system, constituting indeed one of the islets of sclerosis which characterise the affection. On the other hand, when, as is the rule, there is pallor of that portion of the nerve which forms the disc, there is strong evidence in favour of the view that this atrophic condition of this portion of the nerve depends on a patch of sclerosis situated at some portion of the nerve behind the globe, or affecting some portion of the optic commissure or tracts. Sir William Gowers dissents from this view, however; he regards the atrophy in these cases as strictly comparable with that met with in cases of tabes dorsalis; so that instead of regarding the atrophy of the portion of the nerve which forms the disc as secondary to destruction of the nerve at some point behind the globe, he regards it as due to a primary degeneration of the nerve elements. I have elsewhere taken exception to this view, and have pointed out that the morbid changes met with after death in the optic nerves, chiasma, and tracts are strictly in keeping with the random distribution of the lesions seen in other parts of the nervous system in disseminate sclerosis; and that to suppose two portions of the nerve may be affected while an intermediate portion entirely or largely escapes, is quite contrary to the behaviour of any nerve whose elements are undergoing a process of primary degeneration; in this case the degeneration is continuous, and does not affect two or more portions of the length of a nerve-fibre leaving intermediate portions unaffected.

Two classes of cases still remain for our consideration. In one of these, amblyopic defects of vision are complained of by the patient, attended it may be by alterations in the fields of vision on perimetric observation, but in which ophthalmoscopic examination fails to detect any abnormality in the optic discs or other parts of the fundus. Such cases may lead us into grave errors of diagnosis, as the failure on ophthalmoscopic examination to detect any objective cause for the amblyopia may lead us to regard the case as one of hysteria; whereas the amblyopia may depend on a patch of sclerosis situated in some part of the nerve or tracts behind the globe,—a condition that has not existed long enough to lead to secondary atrophy of the termination of the part of the nerve which forms the optic disc. The other class of cases consists of those in which, though distinct pallor of the disc is obvious on ophthalmoscopic examination, there is normal acuity of vision. Though subsequently amblyopic defects of vision may be met with in such cases, it is wonderful for how many years the detection of such a pallor may precede defective vision. Indeed, in the absence of all other signs of disseminate sclerosis the true significance of the pallor of the optic discs may not be appreciated.

All these disturbances of vision have been traced to changes in the optic nerves and tracts; no sclerotic or other changes in the retina have ever been described in this disease.

Besides the defects of vision, affections of the ocular muscles occur; they consist either in nystagmus (see art. "Medical Ophthalmology," vol. vi.), or in paralysis of one or more of these muscles. In the type of the disease portrayed by Charcot and earlier writers, and regarded until the present time as the most common, nystagmus is one of the leading features; but we now know that a very large number of otherwise typical instances of disseminate sclerosis occur in which nystagmus forms no part of the clinical picture. Nevertheless, this symptom occurs in about half the number of cases of the disease, according to Charcot, and in 70 to 80 per cent according to Marie; while Uhthoff found it in 58 per cent of his cases, and Krafft-Ebing in 60 per cent of his. The nystagmus is usually bilateral, but in rare cases it may be present in one eye only. Most commonly it occurs only when the eyes are moved; but it is present sometimes apart from any attempts at voluntary movement of the eyes, and consists, as a rule, in short horizontal jerks to which some vertical or rotatory tendency may be added. It becomes intensified on turning of the eyes to either side, though this increase may be more marked when the eyes are turned in one direction than in the other. Similarly, attempts at fixation as an object is brought nearer to the patient intensifies it; and, like other voluntary movements of the eyes, it may evoke nystagmus when in the resting condition of the globes it is not present; or an upward or downward movement of the eyes may bring the symptom about, in the latter case, however, but rarely; such phenomena we are in the habit of calling "nystagmoid" jerks or movements, as opposed to the spontaneous nystagmus which requires no such devices to call it forth. Nystagmus may only occur on turning of the eyes laterally to one side; or, although it may occur when turned in either direction, the movement to one side may evoke a greater degree of unsteadiness than movement in the opposite direction. We must not mistake for the nystagmus of multiple sclerosis the slight nystagmoid jerks on extreme lateral turning of the eyes to one side which are commonly met with, quite apart from organic disease of the nervous system, in debilitated states such as convalescence from some severe illness; and in my experience it is not uncommon to meet with a similar state of things some years after a myelitis which, at the time of its occurrence, gave rise to no symptoms other than those which commonly result from a transverse lesion of the middle or lower thoracic cord.

Then again Kunn has described a form of nystagmus-like muscle disturbance which he calls "Einstellungszittern," in which there is no nystagmus while the eyes are parallel; but on trying to fix a distant or near object some jerks are at once evoked as the eyes at first overshoot the point, coming too near together at one moment and the next moment going too far beyond the fixation point before finally getting into the right position.

Nor shall we mistake the slight jerky tremulousness of the globes, seen on lateral deviation in hysterical cases, for the nystagmus of organic disease. The functional form of the condition is commonly associated with a blinking movement of the lids; this combination is very characteristic, and should prevent all chance of error.

Ocular paralysis may be actually present when the patient comes under observation; or it may form part of the previous history, and have been recovered from; thus resembling the state of things met with in tabes. Uhthoff found marked paralysis of ocular muscles in 17 per cent of the cases he examined; and in half of these the character of the paralysis pointed to its nuclear origin. Isolated paralysis of the sixth nerve was more commonly met with than similar affection of the third. The power of conjugate movement of the eyes to one side, or of convergence, are most frequently impaired or sometimes lost. When single nerves are involved the sixth is most commonly affected on one or both sides; less frequently there is progressive paralysis of the third, or occasionally the fourth may give evidence of defect.

The pupils are usually equal; but sometimes one is larger than the other, or such inequality may only appear on accommodation. Failure of the pupil to react on accommodation is more commonly met with than loss of its reaction to light; but both conditions are of exceptional occurrence.

Sensory symptoms.—Although sensory defects are not so prominent as the motor, in reality they are rarely absent in some form or other. The most common disturbance of this kind is a subjective feeling of giddiness, which may be one of the earliest symptoms of the affection, and in which it appears to the patient that he and the surrounding objects are both going round. Such a state of things may persist for long periods, or may only occur more or less in paroxysms. Patients so affected often find that they remain free from giddiness as long as they can keep still; but that any movement, even such as putting out the arm to reach a book at the side of the bed, will bring on an attack, which, however, passes off after they have remained quiescent for a little while.

Headache usually occurs in paroxysms; and the pain, though as a rule moderate, may be severe, and may be referred to the back of the head and down the neck, or to the vertex, or the forehead. Such pain is sometimes associated with vomiting, and there may be unilateral headache as in hemicrania in some cases.

Of cutaneous sensory disturbances paræsthesiæ are the most common, and may affect any part of the body, including the areas of supply of sensory cranial nerves; though the hands and feet are most commonly affected. The mucous membranes may participate in these defects. Probably no subjective feeling is more common than that of numbness, which, though sometimes associated with objective blunting of sensibility, commonly exists without the possibility of detection. Feelings of pins and needles, or as if the part had gone to sleep, are common; and various feelings of heat and cold occur. Though such sensations give rise to discomfort, this is in no way comparable to the distress which is sometimes occasioned by cramp-like feelings, which are likened by some patients to a slow twisting and pulling asunder of the parts of the limb on a rack. The concomitance of the most marked feelings of numbness in the same limb with those excruciating paroxysms of cramp-like pains gives rise to mixed feelings of the most distressing character. Sometimes pain in the spinal column is complained of, and I have a patient at present under observation who suffers severe pain in the groin; walking always evokes it, but it disappears after the patient has rested for a short time; the pain, however, comes on spontaneously from time to time quite apart from movement of the limb. Girdle feeling is rare, so are the lancinating pains and gastric crises of tabes.

Objective defects of sensibility are said to be rare, and true it is, as I have said, that with the most pronounced subjective sensations, even of numbness, no blunting of sensibility may be detected. It is probable, however, that such objective defects of sensibility are oftentimes missed as they are so fleeting; so that, although not present on one examination, repeated systematic examinations may prove their existence from time to time in the course of the disease. It is no uncommon experience to find most marked anæsthesia and analgesia in some part one day, and to find no trace of it on the next; and so on. I have met with slight blunting to painful impressions where tactile sensibility appeared to be quite normal. All forms of sensibility may be affected, including an inability to distinguish heat from cold; and these abnormalities, like the paræsthesiæ, are most commonly found affecting the hands and feet. Permanent anæsthesia may occur. Hemianæsthesia is sometimes met with, and may be a hysterical manifestation added to the organic disease; but, on the other hand, permanent anæsthesia of the kind may depend on a patch of sclerosis in the fillet interrupting the sensory path to the brain on one side. Freund found sensibility affected in twenty-one out of thirty-three cases examined; in fourteen of these the defect was transitory, while in the others it was lasting.

Muscular sense may be affected, apart from the existence of cutaneous anæsthesia or in conjunction with it; all notion of the whereabouts of a limb or the position of its component segments being lost. Disturbances of this kind were present in twenty-two of Freund's cases.

Superficial reflexes.—Strümpell attaches importance to the fact that the abdominal reflexes were absent in 67 per cent of 24 cases of disseminate sclerosis; while out of 185 persons with normal nervous systems they were only absent in 13½ per cent. In Probst's series these reflexes were not obtained in 73 per cent of the cases.

Trophic disturbances.—Though such defects are not frequently met with in multiple sclerosis, some degree of vaso-motor disturbance is more common than is generally supposed. Of ten cases of erythromelalgia recently recorded by Dr. James Collier in the *Lancet* of 13th August 1898, no fewer than five and possibly six of the patients were the subjects of disseminate sclerosis. Comparatively early in the course of the disease œdema of the feet and ankles may be met with, which may persist to a slight extent even when the limbs are in the horizontal position; or they may then disappear and only reappear after these parts have been dependent for a time. Swelling of joints may occur, especially the small joints of the hands and feet; and bleb-like blisters may occur in the skin. There may be abnormal local depression of surface temperature as of a limb, and this may be attended with sweating. Trophic disturbances of so severe a character as to lead to the danger of bedsores is only a part of the final stages of a protracted case in which there is usually loss of control over the sphincters; a condition which has played no unimportant part in bringing about this state of affairs.

The nails may become brittle and crack; and the hair is said to fall out in some cases.

Muscular atrophy is one of the trophic defects met with in this disease; but it is only met with in a small group of cases. Such atrophy may involve many muscles, but is more commonly limited to certain groups, especially the small muscles of the hand.

Bladder and rectal troubles.—Quite early in the course of multiple sclerosis there may be some difficulty in passing water; and, according to Oppenheim, the majority of cases present some slight degree of vesical and rectal trouble. Any marked interference with these functions is, however, rare except late in the course of the disease, when there may be complete loss of control over one or both sphincters. When disorders occur early they present a markedly remittent character; and there may be retention or incontinence of urine without any affection of the anal sphincter.

Sexual disturbances.—Increased excitability of the sexual functions may be met with; but some degree of weakness or loss of sexual power is more common, and may occur comparatively early in the course of the disease.